EXHIBIT C33

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**MDL NO. 16-2738 (FLW) (LHG)**

EXPERT REPORT OF MICHAEL BIRRER, MD, PHD FOR GENERAL CAUSATION *DAUBERT* HEARING

Date: February 25, 2019

Michael Birrer, M.D., Ph.D.

BACKGROUND AND QUALIFICATIONS

Following is a brief summary of my background, education, medical training, clinical expertise and research activities.

I earned my undergraduate degree at Rensselaer Polytechnic Institute and graduated with a B.S. in Biology. I subsequently was accepted into the Medical Scientist Training Program at the Albert Einstein College of Medicine and completed my M.D. and Ph.D. in 1982 with my principal area of study in microbiology and immunology. I performed a medical internship at the Massachusetts General Hospital (MGH) and subsequently completed a residency in medicine at MGH. I entered the medical oncology fellowship at the National Cancer Institute (NCI) in Bethesda, Maryland and upon completion of that fellowship, performed a postdoctoral fellowship in the laboratory of Dr. John Minna on the molecular genetics of lung cancer. After completing my fellowship, I joined the faculty at the NCI in the Division of Cancer Treatment as an investigator in 1988. Three years later, I was appointed as a senior investigator (with tenure) and established the molecular mechanism section in the Division of Cancer Prevention and Control. Over the next 17 years, I held a number of positions, including member of the Committee for the Protection of Human Subjects, member of the Gynecologic Oncology Tumor Board, member of the Extramural Institutional Review Board (IRB), member of the Clinical Oncology Fellowship Section Committee, Chair of the Gynecologic Oncology Working Group in the Division of Clinical Sciences and Deputy Branch Chief of the Cell and Cancer Biology Branch.

In November 2008, I was appointed Professor of Medicine at the Harvard School of Medicine. I assumed the position of Director of Gynecologic Medical Oncology and the Gynecologic Oncology Research Program at MGH. This program integrated important new discoveries in translational research into clinical trials. In addition, I became the leader of the Dana Farber/Harvard Cancer Center Gynecologic Cancers program, one of 17 research programs in the DF/HCC. It had 76 members in 7 different institutes, more than $12 million in National Institutes of Health (NIH) funding, and 50 active clinical trials.

In August 2017, I became the Director of the University of Alabama at Birmingham Comprehensive Cancer Center. This center was one of the original 8 comprehensive cancer centers designated in the United States in 1971 and has been continuously funded for 46 years. The Center has 410 members and more than $100 million in cancer research funding. Recently, it has been named the O'Neal Comprehensive Cancer Center, with a gift of $30 million.

I am recognized nationally and internationally as an expert in gynecologic oncology. I have published more than 380 peer-reviewed manuscripts, book chapters and review articles. I have served in leadership positions within the greater gynecologic oncology community. I have been the Chair and Chair Emeritus of the Department of Defense Ovarian Cancer Research Program, a program that awards between $10 and $20 million for ovarian cancer research. I have also served as the chair of the Committee for Experimental Medicine of the Gynecologic Oncology Group, the chair of the Gynecologic Cancer Steering Committee and chair of the Translational Science Working Group of the Gynecologic Cancer Intergroup, and most recently chair of the Core Correlative Science Committee of the NCI. I have been a member of the Society of Gynecologic Oncology (SGO), American Society of Clinical Oncology (ASCO),

American Association of Cancer Research and the International Gynecologic Cancer Society (IGCS) for 10-30 years. In this role, I served on the program committees of ASCO, SGO and IGCS.

My own research efforts have focused almost entirely on the molecular genetics of ovarian cancer. My laboratory has characterized the molecular events in the development of ovarian cancer. This has been supported by two Research Project Grants (RO1 grants). RO1 grants are the mainstay of funding for cancer research from the NIH. They are usually in the range of $3-4 million. The laboratory has participated on the only UO1 grant for the early detection of ovarian cancer within the early detection research network (EDRN). UO1 grants are highly collaborative in nature, combining the expertise of several laboratories to tackle a major scientific/clinical problem. This work has involved the molecular characterization of early lesions of ovarian cancer. The results of this study will directly impact our understanding of the origins of this cancer. The laboratory is also a member of the Clinical Proteomic Tumor Analysis Consortium (CPTAC), which is dedicated to the characterization of cancers on the protein level. This effort will characterize the mechanisms for refractory ovarian cancer and involves multiple well-established laboratories with a budget of approximately $7 million. Finally, I was awarded one of the RC4 stimulus grants (scored at 1% of all grants) focused on the genomics of ovarian cancer. These one-time grants provided up to $5 million to answer big questions about clinically important tumors. The sum total of this is approximately $34 million in funding and more than 20 years of effort focused on ovarian cancer research. My laboratory has helped define the molecular events in ovarian cancers of different histologies and tumor grade. We have defined both early and late genomic aberrations and are currently characterizing both early stage tumors and long-term survivors of ovarian cancer. The laboratory is one of the most knowledgeable in the world on the molecular biology of ovarian cancer and how it relates to the early development of the tumor and its clinical characteristics.

I am being compensated at a rate of $400 per hour for my expert work in this litigation and $1,000 per hour for time spent testifying.

Additional information concerning my credentials and scientific training and achievements can be found in my CV (attached as appendix A).

All of the opinions in this report are stated to a reasonable degree of scientific and medical certainty.

OVERVIEW AND SCOPE OF REPORT

I was asked to address the biological plausibility of plaintiffs' theory that the use of cosmetic talcum can cause ovarian cancer.

Biological plausibility is an important factor in the causation analysis because it assesses the etiology and mechanism(s) of a disease. Advanced tools and scientific knowledge enable researchers to more effectively understand the mechanisms of disease and demonstrate pathways from a purported exposure to the disease. Without an understanding of how a purported exposure can cause a disease, there can be no reliable statement of causation.

Part I of this report provides an overview of what is known about the origins of ovarian cancer, detailing recent molecular biology research, which has given us a clearer picture of the origin and progression of epithelial ovarian cancer. Part II addresses plaintiffs' experts' theories with respect to the supposed migration of talc to a woman's ovaries. Part III addresses what is known about talc and its theorized effects on a woman's ovaries. And Part IV addresses the methodological problems with the experiments of Dr. Saed, which we have now learned were funded by plaintiffs' counsel, and why the results of his experiments do not support the biological plausibility of plaintiffs' theory that perineal talc use can cause ovarian cancer.

I. OVARIAN CANCER

Epithelial ovarian cancer (OC) is a major health problem. It affects 22,000 women each year in the United States and produces 15,000 fatalities annually, making it one of the most lethal forms of cancer in women. The high mortality rate of OC is primarily due to its aggressive nature, as a result of which 75% of the diagnoses are at an advanced stage with the clinical presentation of widespread abdominal dissemination. OC therapy includes debulking surgery followed by taxol/platinum chemotherapy. It has been demonstrated that the residual disease after primary debulking surgery has a crucial impact on survivability. However, the limited overall survival is mainly due to the high rate of tumor relapse and the development of chemo-resistant disease.

A paradigm shift has occurred in our understanding of ovarian cancer. Ovarian cancer is no longer considered a single disease, but rather a composite number of unique cancers, characterized by completely different patterns of genomic alterations and different developmental origins.

The major histotypes of ovarian cancer include serous, endometrioid, clear cell and mucinous, while tumor grade extends from well differentiated (grade 1) through poorly differentiated (grade 3).[1] It has been well recognized that these tumors have different microscopic appearances, biologic characteristics and clinical features. Recent molecular discoveries have demonstrated that they are unique tumors with specific activated biochemical pathways. For high grade serous tumors, there is a profound abnormality in DNA repair. In the ordinary course, cells routinely sustain damage to DNA but employ a range of tools to repair such insults, including by causing cells that are beyond repair to die (to be replaced by healthy cells), a process called apoptosis. Serous tumors arise where this repair process is compromised by inactivating mutations in p53 and BRCA1/2. p53 and BRCA1/2 are genes that make sure the DNA is protected and mutations are corrected. Low grade serous cancers of the ovary differ from high grade serous cancers in that they have a high frequency of ras mutations with activation of the MAP kinase pathway and wild type p53. Ras mutations are very rare in high grade tumors while almost all of these tumors have p53 mutations.[2] Endometrioid ovarian cancers have mutations with the PI3 kinase pathway and CTNNB1, which are important genes that tell the

[1] Cannistra, *Cancer of the ovary*. N Engl J Med. (2004) 351(24):2519-29; Cho & Shih, *Ovarian cancer*. Annu Rev Pathol. (2009) 4:287-313.

[2] Cancer Genome Atlas Research Network, *Integrated genomic analyses of ovarian carcinoma*. Nature (2011) 474(7353):609-15.

cancer cell to grow or survive. Clear cell cancers have inactivating mutations in the tumor suppressor gene ARID1A, which is an important gene involved for maintaining the structure of the chromosome. Finally, mucinous tumors have activating mutations to ras, another signaling gene that protects the cell from the effects of chemotherapy.

The importance of these findings is that what we originally called ovarian cancer as a single disease is actually a collection of separate diseases. There is minimal molecular overlap among these tumors, which in part explains their diverse clinical presentations and natural histories. Gene expression profiling has also clearly demonstrated the distinct nature of ovarian tumors in relation to tumor histology and grade. These molecular discoveries have reinforced the view that ovarian cancer is actually a series of separate diseases with unique molecular features and different developmental origins.

It is now clear that a subset of clear cell and endometrioid cancers arise from endometriosis. Pathologic evidence has long showed these cancers in the presence of endometriosis and rare cases have described transition lesions. These cases show pathologic transition between benign endometriotic lesions to abnormal epithelial cells to ovarian cancer. More recent molecular data have confirmed this relationship by showing that the same mutation in ARID1A (an important mutation for the development of clear cell and endometriotic cancers of the ovary) can be found in the tumor and the adjacent endometriotic lesion. It is well accepted that these cancers arise from a process that transforms the endometriotic lesion.

In contrast, serous cancers of the ovary originate from a different tissue source and through a different process. High grade serous ovarian cancers arise primarily from the fallopian tube. Pathologic evaluation of fallopian tubes from prophylactic oophorectomies of BRCA 1/2 germ-line mutated patients revealed early invasive cancers in the fimbria and occasionally serous intraepithelial cancers (STICs). These latter lesions are entirely consistent with a precursor lesion for high grade serous ovarian cancer. Of interest, in addition to STICs, TP53 signatures are also found in the fallopian tubes. TP53 signatures are characterized by small collections of normal appearing fallopian tube epithelium (without any additional pathologic findings, including cellular inflammation), which stain for TP53. These strips of fallopian epithelium contain a mutated p53 gene (resulting in the accumulation of TP53), and it is likely some of these evolve into STICs and then into ovarian cancer.

The modern view of the molecular biology of ovarian cancer described above has important consequences both for our approach to the disease and also future research. Clearly, the molecular abnormalities and the pathways they affect will become important potential therapeutic targets, but also aid in early detection and the identification of prognostic biomarkers. Equally important, research that will move the field forward needs to recognize and incorporate the established features of the tumor described above. **This means utilizing appropriate cell lines and *in vivo* models to fit the research question being tested**. For example, utilizing a clear cell cancer cell line to test questions about HGSOC is of little value. Likewise, testing a particular hypothesis in ovarian cancer without *in vivo* experiments is unlikely to yield important data and meaningful discoveries. In all events, inquiries into the causes of one type of ovarian cancer may tell us nothing at all about the causes of another type of ovarian cancer because of the distinct pathways through which these diseases develop.

II. MIGRATION

A. General Observations

Plaintiffs' experts opine that it is accepted in the scientific community that talc can migrate to the ovaries from the perineum and/or through inhalation. Generally speaking, plaintiffs' experts' reports rely on two lines of supposed evidence to support this assumption: 1) the claimed presence of talc particles in ovaries and/or ovarian cancer tissue; and 2) experimental results attempting to demonstrate the transition of particles from the vagina up to the ovary. A closer investigation of the science, however, shows that plaintiffs' experts are significantly misinterpreting the findings of these articles and exaggerating their importance.

i. Supposed Presence Of Talc In Ovaries

The proposed finding of talc particles in ovarian cancers rests primarily on three publications, all of which have severe limitations. As explained further in this sub-section: (1) the first study used improper methods to identify talc and may have been tainted by contamination; (2) the second study found talc in a lymph node – not ovaries; and (3) the third study found talc in women's ovaries who had **not** used talc perineally and therefore says absolutely nothing about perineal talc use. To conclude based on these three studies that talc reaches the ovaries and therefore can cause ovarian cancer is speculative and highly misleading.

Talc and Carcinoma of the Ovary and Cervix by Henderson et al. (1971)[3] reports the incidence of talc particles in a series of ovarian and cervical cancers. They report that 75% (10/13 patients) of the ovarian cancer tumors had talc particles, as did 5/12 normal ovaries from breast cancer patients. The talc was identified only by electron microscope (EM), which is not the standard analysis done now. The newer standard uses several different technologies, which increases the likelihood of finding rare particles of talc. Thus, it is difficult to credit the talc particle counts and uncertain whether talc was actually more prevalent in the ovarian cancer patients. The paper provides insufficient description as to the techniques used to gather the tissue and process it. Given the high frequency in the control specimens (breast cancer patients), laboratory contamination has to be a major concern. Although this study is cited by at least ten of plaintiffs' experts to support the theory that talc is present in women with ovarian cancer,[4] it does not support their opinions because their methods were not reliable, and any talc may have resulted from contamination. And of course, the paper certainly does not support the theory that such presence of talc – even if true – causes ovarian cancer.

Cramer et al., ***Presence of Talc in Pelvic Lymph Nodes of a Woman with Ovarian Cancer and Long-Term Genital Exposure to Cosmetic Talc*** (2007)[5] reports a single case of a

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[3] Henderson et al., *Talc and carcinoma of the ovary and cervix*. J Obstet Gynaecol Br Commonw. (1971) 78(3):266-72.

[4] Saed Rep. at 12; Smith-Bindman Rep. at 13, 35; Kane Rep. at 13, 14, 16; McTiernan Rep. at 58, 63; Carson Rep. at 4; Clarke-Pearson Rep. at 4, 8; Kessler Rep. at 23; Blair Smith Rep. at 16; Wolf Rep. at 5, 11; Plunkett Rep. at 26, 33, 49, 60; Moorman Rep. at 33; Singh Rep. at 18, 57.

[5] Cramer et al., *Presence of talc in pelvic lymph nodes of a woman with ovarian cancer and long-term genital exposure to cosmetic talc*. Obstet Gynecol. (2007) 110(2 Pt 2):498-501.

woman with ovarian cancer, a history of talc use and the identification of talc within a lymph node. The talc was identified by polarized light microscopy, scanning EM and x-ray spectroscopy. This report adds little support to the migration argument. It is a single case (despite a discussion of 12 other cases, there are no data presented on them), and the talc is found in the lymph node. There are no hypotheses on ovarian cancer arising from a process in the lymph node, and it is difficult to imagine talc reaching the ovary via the lymph system because the lymph system drains the abdomen and does not flow into it. Dr. McTiernan claims this study "demonstrate[s] talcum powder products can migrate from the perineal area to the ovaries and fallopian tube through both genital tract migration and inhalation."[6] A single case report cannot conclude anything and more importantly, it is essentially irrelevant because lymphatic spread is not thought to have any relevance to the origin of ovarian cancer.

The third study may provide the best explanation for the results of the other two. This 1996 study by Heller et al., entitled ***The relationship Between Perineal Cosmetic Talc Usage and Ovarian Talc Particle Burden***[7] is a study of 24 patients who underwent oophorectomies and were interviewed pertaining to talc usage (12 of whom reported high talc use and 12 of whom reported never using talc). Talc particles were detected in the ovaries of all 24 cases regardless of exposure. While the authors explain this finding by implicating diaper use for the source of talc, there is no direct evidence for this at all. This explanation also does not make sense because the mean age of the women in the study was 49, meaning that the talc would have had to travel to the ovaries in their infancy and remain lodged there for decades. By contrast, the finding of talc in non-talc users is plausibly consistent with environmental contamination of operative specimens, either in the operating room or pathology suite. Although the authors mention examining solutions for the presence of talc, there is no detailed description about how they did it and specifically which solutions were tested. There are also many potential sources of talc that could be contaminants and no description of talc control methodology to ensure the operating rooms and pathology suites were free of talc. The evaluation of control tissues is absolutely critical to these types of studies. In this study, there are no controls, i.e., abnormal or normal tissues (other than the female genital tract) that would not be expected to have talc. The presence of talc in such control tissues would further support contamination. Dr. Smith-Bindman (along with several other plaintiffs' experts) cites this study to support the proposition that talc migrates up the fallopian tubes and thereby plays a role in the development of ovarian cancer.[8] As described above, however, talc particles were detected in every case ***regardless of history of exposure***; there is no detailed description about how the authors examined solutions for the presence of talc; and the study lacked any control. If anything, this study suggests that talc found in a woman's ovarian tissue bears no relation to perineal talc use.

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[6] McTiernan Rep. at 59. At least eight other experts also rely on this study. Saed Rep. at 12; Kane Rep. at 4, 14; Siemiatycki Rep. at 65; Wolf Rep. at 11; Zelikoff Rep. at 14; Plunkett Rep. at 29; Moorman Rep. at 33; Singh Rep. at 18, 20, 57.

[7] Heller et al., *The relationship between perineal cosmetic talc usage and ovarian talc particle burden*. Am J Obstet Gynecol. (1996) 174(5):1507-10.

[8] Smith-Bindman Rep. at 35. Several other experts likewise rely on this study for that proposition, including Kane Rep. at 14, 30; McTiernan Rep. at 29, 58, 59); Carson Rep. at 6; Clarke-Pearson Rep. at 8; Siemiatycki Rep. at 65; Wolf Rep. at 11; Zelikoff Rep. at 19; Plunkett Rep. at 28, 29, 35); Moorman Rep. at 33; Levy Rep. at 13, Singh Rep. at 18, 20, 57.

ii. Hypothesized Migration of Talc to Ovaries

The second line of evidence relied on by plaintiffs' experts are studies that supposedly support the theory that talc applied perineally can enter the vagina, travel through the cervix and endometrium and then travel up through the fallopian tube to the ovary. Not surprisingly, there are no studies that validate this theory, which is contrary to basic anatomy and common sense. Instead, the studies relied on by plaintiffs' experts either did not involve talc and/or involve the actual insertion of materials inside a woman's body, rather than dusting the outside of her body. These include the following:

1. Venter et al., ***Migration of a particulate radioactive tracer from the vagina to the peritoneal cavity and ovaries*** (1979).[9] (This study is cited by the following plaintiffs' experts: Smith-Bindman (page 35), Kane (page 14), McTiernan (pages 58, 59), Carson (page 7), Clarke-Pearson (page 8), Siemiatycki (page 65), Wolf (page 11), Zelikoff (pages 12, 13), Plunkett (pages 28, 31).) The authors studied the migration of radionucleotide labelled human albumin microspheres as a model for talc in humans. They report 9 out of 21 patients demonstrated radioactivity in tubes and ovaries at the time of surgery. This study has serious methodologic flaws. First, the authors used the Tc-labelled human albumin microspheres as a surrogate for talc. It is well known that the radiolabels can disassociate from the protein (albumin) or that the protein can break down. Thus, the tracer (which produces the radioactivity) can travel separately and extensively compared to the whole complex. There are no controls for this at all in the study. Second, the study design necessitated putting the patients in a supine (lying-down) position with the buttocks slightly elevated and after injection kept like this for 2 hours with the legs pressed together. This is obviously not equivalent to dusting the groin with powder in a woman in the vertical position. Finally, it is impossible to determine from the study the exact level of radioactivity detected in the organs or for that matter, the blood. There are no numbers provided. Plaintiffs' experts' reliance on this study is flawed because the study does not eliminate the possibility that the radioactivity has broken off from the larger particle and traveled through the body in a mode completely different from direct migration up the fallopian tube. Moreover, there are no data in this study as to the frequency of disassociation of the isotope for albumin, its circulation or its distribution within the body.

2. Sjösten et al., ***Retrograde migration of glove powder in the human female genital tract*** (2004).[10] (This study is cited by Smith-Bindman (pages 1, 35), McTiernan (page 59), Carson (page 7), Clarke-Pearson (page 8), Blair Smith (page 16)), Wolf (page 11), Plunkett (pages 28, 36), Zelikoff (page 12), Moorman (page 33) and Singh (pages 18, 19, 20, 57).) This study reports increased glove powder in the cervix, tubes and uterus in patients who had been exposed to gloves with powder compared to those exposed to gloves without powder. Although Dr. Clarke-Pearson, for example, cites this study as

[9] Venter & Iturralde, *Migration of a particulate radioactive tracer from the vagina to the peritoneal cavity and ovaries*. S Afr Med J. (1979) 55(23):917-9.

[10] Sjösten, Ellis & Edelstam, *Retrograde migration of glove powder in the human female genital tract*. Hum Reprod. (2004) 19(4):991-5.

supporting his retrograde migration theory, there are important and serious limitations of this study in relation to the present litigation. First, the study involved starch, not talc preparations. There are well documented differences both in physical size and biochemical properties between talc and starch that make these comparisons irrelevant. Second, the delivery of starch particles in this set of experiments is from bimanual exams using powdered gloves. This approach delivers the foreign bodies at the cervical OS with considerable intravaginal pressure, which, again, is quite different from perineal dusting.

3. Egli et al., ***The transport of carbon particles in the human female reproductive tract*** (1961).[11] (This study is cited by Zelikoff (page 13), Smith-Bindman (page 35), Kane (page 14), McTiernan (page 58), Carson (page 7), Clarke-Pearson (pages 7, 8), Blair Smith (page 16), Wolf (page 10), Zelikoff (page 13), Plunkett (pages 28-30) and Singh (pages 15, 60).) This study researched the mechanism by which spermatozoa reaches the oviduct in mammals by using carbon particles in humans as a model. A solution of dextran and bone black (carbon particles) was deposited into the distal vagina near the cervix in anesthetized women in the lithotomy position while an intramuscular injection of oxytocin was given. The patients then underwent the planned surgical procedure and the removed tubes were flushed with saline in an attempt to detect the carbon particles. Contrary to plaintiffs' experts' reliance on this study, there are many aspects of this study that limit its relevance and scientific value. First, there are no data presented to ensure that carbon particles are equivalent to talc in size and physical properties. Second, the patients were injected with oxytocin, a hormone known to induce muscular contraction within the female genital tract prior to general anesthesia. These are not conditions consistent with genital dusting in a standing female. Dr. Ellen Blair Smith emphasizes that "no propulsive force of talc was used in the study."[12] But this emphasis ignores the fact that carbon particles were placed in the posterior fornix of the vagina while the woman was under anesthesia, in the lithotomy position, and before being injected with oxytocin. Such a highly contrived model system tells us nothing of relevance to the dusting of the perineum with powder.

4. Kunz, et al., ***The Uterine Peristaltic Pump*** (1997).[13] (This study is cited by Saed (page 12), Wolf (page 11) and Plunkett (pages 28, 35,36, 37); it was also considered but not cited by Carson (ex. B), Clarke-Pearson (ex. B), Blair Smith (ex. C), Zelikoff (ex. B), Levy (ex. B) and Smith-Bindman (reliance list).) The first part of this study utilizes vaginal sonography to detect uterine contractions during the female menstrual cycle. This obviously measures changes in the uterine wall (via contractions) with an indirect interpretation of endometrial cavity pressure and therefore is not a direct measurement. In addition, there are no data presented to determine the direction of the pressure generated by the uterine wall contraction. Further, the act of transvaginal manipulation itself can certainly affect uterine contraction activity. That is assumed from the results of the

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[11] Egli & Newton, *The transport of carbon particles in the human female reproductive tract*. Fertil Steril. (1961) 12:151-5.

[12] Smith Rep. at 16.

[13] Kunz et al., *The uterine peristaltic pump. Normal and impeded sperm transport within the female genital tract*. Adv Exp Med Biol. (1997) 424:267-77.

second part of the study. The second methodology used was hysterosalpingoscintigraphy utilizing technetium-labelled albumin macrospheres of the same approximate size of human spermatozoa. Obviously, there are major differences between the macrospheres and human spermatozoa, not the least being the motility of spermatozoa and its entirely different biochemical composition, as described above. This study has a series of problems, including the use of radiolabeled albumin spheres, which can dissociate from the isotope, and the fact that the placement of these spheres into the reproductive tract of women is fundamentally different from dusting the perineum.

B. Additional Methodological Flaws In Plaintiffs' Experts' Opinions Regarding Migration Of Talc To The Ovaries

There are several additional methodological flaws in plaintiffs' experts' opinions regarding the migration of talc to the ovaries.

Dr. Clarke-Pearson – Dr. Clarke-Pearson analogizes to the migration of sperm into the tubes after coitus. It is rather surprising to hear this from a gynecological oncologist. The obvious difficulty with this line of reasoning is the fact that spermatozoa are motile and have evolved over millions of years to be able to migrate under their own control to increase the potential to fertilize the egg. This mode of transport is not consistent with a talc particle. Further, it should not need to be pointed out that the sperm is being delivered with considerable force and pressure at the cervical OS during the act of coitus rather than dusting on the surface of the perineum.

Dr. Smith-Bindman – In addition to relying on the studies set forth above, Dr. Smith-Bindman also claims that epidemiology data showing a reduced risk for ovarian cancer in women who underwent a tubal ligation is evidence for the role of talc in the development of ovarian cancer. This opinion, too, is not supported by the science. I note at the outset that while Dr. Smith-Bindman repeatedly asserts that studies show that the risk of ovarian cancer in talcum powder users is reduced in women who have undergone tubal ligation and hysterectomy,[14] she does not cite any studies that support this claim.

There are studies that suggest a ***generally*** protective effect of tubal ligation, but there is no evidence that this effect is related to the blockage of transition of some agent through the fallopian tube. In fact, a number of studies have concluded that tubal ligation and/or hysterectomy has no effect on ovarian cancer risk in women who use talc perineally. For example, a study by Gertig et al. – the only cohort study that addressed the effect of tubal ligation and/or hysterectomy and the occurrence of ovarian cancer – concluded that "no effect modification was seen by history of tubal ligation."[15] A pooled study by Terry et. al. found that "exposure to genital powder applications that occurred before tubal ligation or hysterectomy

[14] Smith-Bindman Rep. at 15, 35.

[15] Gertig et al., *Prospective study of talc use and ovarian cancer*. J Natl Cancer Inst. (2000) 92(3):249-52.

made no substantive difference in the results."[16] There have also been case-control studies that have concluded that there was a lower incidence of ovarian cancer in talc users who had tubal ligation, but not for patients who had hysterectomies. For example, Cramer et al. (1999) found odds ratios of 0.98 and 1.80 for tubal ligation/no tubal ligation and 2.61 and 1.60 for hysterectomy/no hysterectomy[17]; and Mills et al. noted odds ratios of 0.88 and 1.54 for tubal ligation/no tubal ligation and odds ratios of 1.79 and 1.33 for hysterectomy/no hysterectomy.[18] This is inherently illogical, since either procedure would cut off plaintiffs' theorized pathway for talc migration. As such, the support for a theory by which the protective effect of tubal ligation can be attributed to shielding the ovaries from talc is lacking.

Notably, recent data have demonstrated that there are dramatic effects on the cells at the distal end of the fallopian tube cells after a tubal ligation.[19] Given the role of the fimbria in the development of the majority of ovarian cancers, the demonstration of substantial development of quiescence cells in this region is highly relevant. It is likely that these cells cannot be transformed into cancer cells in this state. Thus, the effects of tubal ligation are likely related to biologic effects on the epithelial cells within the fallopian tube, which in turn decreases the number of cells that are potential precursors for ovarian cancer – and not to the elimination of a pathway for cancer initiators or promoters that ostensibly travel up the fallopian tubes toward the ovaries.

Dr. Wolf – In addition to the studies discussed above, and the sperm analogy, which is contrary to basic human biology, Dr. Wolf also invokes retrograde menstruation as evidence that talc could travel to the ovaries.[20] But retrograde menstruation is a different process; it occurs more closely to the fallopian tubes than does perineal dusting, and it may be facilitated by uterine contractions that would not be occurring in the ordinary course during perineal dusting. In addition, menstrual fluid contains blood and endometrial cells, which is very different from talc particles.

Dr. Zelikoff – Dr. Zelikoff goes even further down the path of speculation, opining that ultrafine particles can migrate from the respiratory system to the systemic circulation.[21] As shown below, the studies she relied on did not use talc, explored the effects of particles on rats, rabbits or other animals with dissimilar anatomy to humans, involved dissimilar exposure conditions, including high doses and direction installation or ***injection*** of the particulate into the body, and did not study particle migration to the reproductive system or the translocation theory

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[16] Terry et al., *Genital powder use and risk of ovarian cancer: a pooled analysis of 8,525 cases and 9,859 controls*. Cancer Prev Res (Phila). (2013) 6(8):811-21.

[17] Cramer et al., *Genital talc exposure and risk of ovarian cancer*. (1999) 81(3) Int J Cancer. 351.

[18] Mills et al., *Perineal Talc Exposure and Epithelial Ovarian Cancer Risk in the Central Valley of California*. (2004) 112 Int'l J. Cancer 458.

[19] Tiourin et al., *Tubal Ligation Induces Quiescence in the Epithelia of the Fallopian Tube Fimbria*. Reprod Sci. (2015) 22(10):1262-71.

[20] Wolf Rep. at 10.

[21] Zelikoff Rep. at 14-17.

pertaining to the reproductive system.

- Werebe, et al.'s study, ***Systemic distribution of talc after intrapleural administration in rats***,[22] injected high doses of talc slurry directly into the pleural cavities of rats, and found talc particles throughout organs, including the chest wall, lungs, heart, brain, spleen and kidneys, of rats 24-48 hours after injection. This study has limited applicability to the question of talc in humans because it was performed on rats, involved direct application into the pleural cavity, and the authors did not look at whether talc was found in the reproductive system.

- Driscoll et al., ***Effects of particle exposure and particle-elicited inflammatory cells on mutation in rat alveolar epithelial cells***,[23] explored the intratracheal installation in rats of quartz, carbon black and titanium dioxide at levels eliciting a neutrophilic inflammatory response found to increase mutation of cells in lungs. This study involved rats, did not involve talc, used direct installation of particulate into the body (not perineal application), involved dissimilar dose exposure conditions, did not look at the reproductive system and did not explore any translocation theory.

- In Ferrer et al.'s article, ***Influence of particle size on extrapleural talc dissemination after talc slurry pleurodesis***,[24] high doses of two different sizes of talc were injected into the pleural cavities of 20 rabbits. The authors observed greater systemic talc particle deposition of smaller sized talc particles in the lungs, chest wall, diaphragm, mediastinal pleura, heart, liver, spleen and right kidney 24 hours and 7 days after exposure. The authors also noted greater inflammation with talc particles of a smaller size. Importantly, the trends noted were not consistent across rabbits or locations. For example, after 24 hours, no talc was found deposited in the liver for any rabbits, but after 7 days, only 3 of the 5 rabbits demonstrated talc in the liver. And although 1 out of 5 rabbits had talc depositions in the kidney after 24 hours, no rabbits had talc in the kidney after 7 days. These inconsistencies render the analysis of this article impossible to interpret. Moreover, the relevancy of this study is also limited as the study involved rabbits, used direct injections, involved dissimilar dose exposure conditions, did not look at the reproductive system or how the translocation theory applies to the reproductive system and utilized small sample sizes. There was no direct evaluation of reproductive tissues to determine whether any talc particles were deposited in them. It should be noted that in pleurodesis in human patients, there is no reported increase in ovarian cancer nor the presence of talc in the reproductive organs.[25]

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[22] Werebe et al., *Systemic distribution of talc after intrapleural administration in rats*. Chest. (1999) 115(1):190-3.

[23] Driscoll et al., *Effects of particle exposure and particle-elicited inflammatory cells on mutation in rat alveolar epithelial cells*. Carcinogenesis. (1997) 18(2):423-30.

[24] Ferrer et al., *Influence of particle size on extrapleural talc dissemination after talc slurry pleurodesis*. Chest. (2002) 122(3):1018-27.

[25] Viskum K, *et al*. *Long term sequelae after talc pleurodesis for spontaneous pneumothorax*. Pneumologie. (1989) 43:105-6.

- Genofre et al., in a study entitled ***Talc pleurodesis: evidence of systemic inflammatory response to small size talc particles***,[26] injected high doses of two different sizes of talc – small (1.6-7.3 µm, mean: 6.41 µm) or mixed (6.4-50.5 µm, mean: 25.4 µm) particles – in the pleural cavities of 30 rabbits. The authors observed acute systemic inflammatory response for both small and mixed talc injection groups, but "small particle talc produced a more pronounced pleural and systemic response and resulted in greater particle deposition in the organs than mixed talc." The particles found in the organs were smaller than 5 µm, and a significantly larger number of talc particles were observed in both lungs, the liver and kidneys in the small particle talc group compared to the mixed talc group, whereas no significant difference was observed for the spleen. The authors stated that the data did not permit a conclusion as to whether systemic cellular response was due to flow of cells from "pleural cavity to the bloodstream or to a direct system cellular response to the presence of talc particles in the organs," but they speculated that "intense pleural inflammation caused by talc promotes the loss of integrity of the pleural barrier, permitting the free flow of cytokines and talc particles between the two compartments." The relevancy of this study is limited as the study involved rabbits, used direct injections, involved dissimilar dose exposure conditions and did not look at the reproductive system or how the translocation theory applies to the reproductive system.

- Hollinger, ***Pulmonary toxicity of inhaled and intravenous talc***,[27] merely compiles a handful of studies purportedly showing that talc can be accidentally inhaled or injected as part of drug abuse and studies purportedly showing that talc particles trapped in the lungs can induce foreign body granulomas and pulmonary fibrosis. The article does not present any new findings or data and is limited because it does not address translocation of talc outside of the lungs and involves exposure conditions dissimilar to perineal talc application, including dose.

- Kreyling et al., ***Ultrafine particle-lung interaction: Does size matter?***,[28] reviewed existing literature on inhalation of insoluble ultrafine particles inhaled and systemic translocation. No talc studies were considered by the authors and the analysis was limited to inhalation and respiratory system studies, with no consideration of translocation to the reproductive system.

- Nakane's ***Translocation of particles deposited in the respiratory system: a systematic review and statistical analysis***[29] is a systematic review and statistical analysis of previous reports on particle translocation from the respiratory system. Although the article found that particle size was a strong factor for translocation, the authors did not consider any

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[26] Genofre et al., *Talc pleurodesis: evidence of systemic inflammatory response to small size talc particles*. Respir Med. (2009) 103(1):91-7.

[27] Hollinger, *Pulmonary toxicity of inhaled and intravenous talc*. Toxicol Lett. (1990) 52(2):121-7.

[28] Kreyling, Semmler-Behnke & Möller, *Ultrafine particle-lung interaction: Does size matter?* J Aerosol Med. (2006) 19(1):74-83.

[29] Nakane, *Translocation of particles deposited in the respiratory system: a systematic review and statistical analysis*. Environ Health Prev Med. (2012) 17(4):263-74.

talc studies and limited translocation to the respiratory system, with no consideration of translocation to the reproductive system. The authors also noted that their analysis was hindered by a deficiency of information, information bias, publication bias and non-consideration of differences in structures and fabrications of different particles. The relevance of this review to the role of talc in the development of ovarian cancer remains very unclear.

- The Peters et al. study, entitled ***Translocation and potential neurological effects of fine and ultrafine particles a critical update***,[30] addresses particulate air pollution and its association with cardiovascular and neurodegenerative effects and summarizes evidence pertaining to mechanisms involved in the translocation of particles from the lung to other organs. The authors state that their work demonstrates particles can be translocated to other organs by circulating blood, although they caution that "it remains to be shown by which mechanisms ultrafine particles penetrate cellular membranes by non-specific means." The authors also note that some studies did not demonstrate translocation to the lung from other organs. The relevancy of this study is further limited as the article did not include any talc studies and was limited to translocation from the respiratory system with no discussion of translocation to the reproductive system.

- In Rossi's et al.'s ***Acute inflammatory response secondary to intrapleural administration of two types of talc***.,[31] 100 rabbits received intrapleural injections of large quantities of two different sizes of talc. The authors reported increased pulmonary and inflammatory response from talc particles, with greatest effects seen from the smaller talc particles. However, the authors observed no difference in the number of talc particles in the lungs between the control and test groups and did not observe an increased inflammatory response in talc-injected subjects with all parameters. Additionally, the relevancy of this study is also limited as the study involved rabbits, used direct intrapleural injections, involved dissimilar dose exposure conditions, did not look at the reproductive system or how the translocation theory applies to the reproductive system and utilized small sample sizes.

Dr. Levy – Dr. Levy appears to posit that talc could produce an inflammatory "environment" that could contribute to the development of ovarian cancer even without reaching the ovaries through "secondary effects" from unidentified "neighboring or surrounding tissues."[32] But as Dr. Levy acknowledged, this theory is "uninvestigated," and he is "not aware of any studies that have made that delineation of talc exposure to neighboring or surrounding organs."[33] Neither am I, and Dr. Levy's unsupported musings certainly do not provide scientific evidence that talc can migrate sufficiently far from the perineum to produce an effect on the

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[30] Peters et al., *Translocation and potential neurological effects of fine and ultrafine particles a critical update*. Part Fibre Toxicol. (2006) 8;3:13.

[31] Rossi et al., *Acute inflammatory response secondary to intrapleural administration of two types of talc*. Eur Respir J. (2010) 35(2):396-401.

[32] Levy Dep. 165:2-166:12.

[33] Levy Dep. 165:10-16.

ovaries, either directly or by "secondary effects."

III. STUDIES ON THE BIOLOGIC EFFECTS OF TALC ON OVARIES

Studies on the biologic effects of talc on the ovarian epithelium have been limited in number and in general are of poor quality. In fact, the best study to date on the *in vivo* effects of talc on the rat ovary is Hamilton et al., ***Effects of Talc on the Rat Ovary***,[34] which undermines plaintiffs' theories because exposure to high amounts of talc did not result in the development of ovarian cancer. Several of plaintiffs' experts cite Hamilton for the proposition that talc exposure leads to "adverse effects" on rat ovaries (e.g., McTiernan Rep. at 62; Wolf Rep. at 12; Plunkett Rep. at 26, 39), but in so doing, they miss the main point of the study, which is that none of the rats developed ovarian cancer.

Below, I address the studies relied on by plaintiffs' experts, the Hamilton study and plaintiffs' various theories regarding "inflammation."

A. Buz'Zard

Several of plaintiffs' experts rely on a study by Buz'Zard et al. entitled: ***Pycnogenol reduces Talc-induced Neoplastic Transformation in Human Ovarian Cell Cultures***,[35] which reported that talc increases the cellular proliferation of normal ovarian epithelial cells and a granulosa cell line, induces cellular neoplastic transformation and generates reactive oxygen species in cell culture.[36] To the extent these experts rely on this study to support the conclusion that talc exposure can cause ovarian cancer, that is wrong for a number of reasons.

First, the authors utilized a granulosa cell line, which is irrelevant to epithelial ovarian cancer. In addition, the "normal" ovarian cells tested were not normal; rather, they were immortalized. Although the method is not described in this paper, it is likely they were immortalized by SV40 (large and small T), which means they can grow in 3D cultures and by definition are not normal. Normal cells are critical to these types of studies because they will determine the true effects of talc.

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[34] Hamilton et al., *Effects of Talc on the Rat Ovary*. Br J Exp Pathol. (1984) 65(1):101-6.

[35] Buz'Zard & Lau, *Pycnogenol reduces talc-induced neoplastic transformation in human ovarian cell cultures*. Phytother Res. (2007) 21(6):579-86.

[36] For example, Dr. Siemiatycki cites this study for the proposition that "[a]lternative plausible mechanisms of carcinogenicity include talc induced oxidative stress." (Siemiatycki Rep. at 65.) Similarly, Carson portrays this study as showing that "[t]alcum powder caused proliferation of human ovarian cells in culture, and causes these cells to express reactive oxygen species." (Carson Rep. at 5.) And Kane claims that this study is evidence "that talc causes neoplastic transformation in ovarian cells." (Kane Rep. at 36.) Several other experts also rely on this study for similar propositions. (Singh (page 19), Plunkett (page 42), Zelikoff (page 25), Wolf (page 12), Blair Smith (page 17), Clarke-Pearson (page 4), McTiernan (page 60) and Levy (page 14).)

Second, the proliferation assays are difficult to interpret due to the fact that they are viability assays, which only indirectly measure cell number. It is critical that they directly count cells to ensure cellular proliferation has increased.

Third, the effects of talc are minimal, time-dependent and divergent depending upon dose. For example, an increase in the viability assay seen at 24 hours disappears at 72 hours, a result that is difficult to rationalize. Are the control cells growing faster at 72 hours or are the treated cells starting to die? Certainly, the talc remains in the culture fluid. This seems very hard to interpret along with the opposite effects depending upon the talc concentration. Specifically, high concentrations result in inhibition on control cells, while lower concentrations are reported to be stimulatory – an anomalous result. There is no data to explain any of these results.

Fourth, the only measure of cellular transformation is soft agarose growth. It is well known that this assay can provide misleading results in that many human tumors do not grow in these conditions and non-transformed cells can. In fact, the OSE2a cells used in these experiments are likely an example of non-transformed cells, which grow in soft agarose. In figure 2 of the paper, the OSE2a cells demonstrate low but nevertheless cloning ability. Thus, these cells are already able to grow in 3D without any talc exposure – meaning that their growth does not signify malignancy. A much more accurate measure of neoplastic transformation is tumor formation in immunodeficient mice. This is a generally accepted and standard assay for determining whether cells are malignant. But it was not done in this study. Thus, the measure of increased 3D growth after talc treatment is problematic in that the untreated cells are already cloning, and more importantly, this is not a measure of transformation.

Fifth, the paper reports that "*talc caused an initial dose-dependent decrease in ROS [Reactive Oxygen Species] generation which increased with time in OSE2a cells.*"[37] This is not the case. All doses and time points except one (50ug/ml at 120 hours) remained below the control levels. Talc does not increase ROS production in this system; to the contrary, the general effect of talc was to *decrease* ROS relative to controls, and it was this effect that changed over time.

Sixth, there are no controls in this study, such as non-talc particles of approximately the same size (glass beads). This is critical, as it would help determine whether any effects of exposure were attributable to talc specifically, or rather the physical size and shape of the particles (in which case any effects of exposure would not be specific to talc).

Seventh, the effects of pycnogenol are irrelevant to this litigation. This is an experimental agent, which has no approved medical indications. Its effects on ovarian cancer cells are irrelevant to questions about talc.

In short, the Buz'Zard study does not provide a reliable basis for plaintiffs' experts' inflammation hypothesis because, among other things, it used an irrelevant cell line, the effects of talc treatment are conflicting and difficult to interpret and no controls were used. Of further note, the article was published in 2007, and yet even today there is no general acceptance of the notion that talc use fosters an inflammatory process that leads to ovarian cancer.

[37] Buz'Zard & Lau (2007).

B. Shukla

Another study cited by a number of plaintiffs' experts is ***Alterations in gene expression in human mesothelial cells correlate with mineral pathogenicity*** by Shukla et al.[38] For example, Dr. Plunkett cites this study to argue that the available *in vitro* and animal study data show that there is a dose-response relationship for talc toxicity.[39] This study does not support plaintiffs' experts' conclusions because it addresses gene expression – which cannot by itself tell us anything about the ostensible carcinogenicity of talc – and focuses largely on mesothelial cells and asbestos and, to the limited extent it addressed talc and ovarian cells, it showed no effect on gene expression.

The Shukla study reports the effects of low or high concentrations of crocidolite asbestos, nonfibrous talc, fine titanium dioxide or glass beads on immortalized mesothelial cells and human ovarian epithelial cells. Its primary endpoint was on gene expression levels. While gene expression is a molecularly interesting endpoint, its precise biologic effect and impact can be quite complex and difficult to predict. Many physical and biochemical stimuli can alter gene expression patterns without an obvious resulting biologic change. Distinguishing a real effect from background regulatory noise can be very difficult. The changes seen in this paper are relatively small in amplitude and in number. Validation by both real time Polymerase Chain Reaction and protein evaluation, along with careful biologic experiments, would be required to conclude that the changes are relevant.

This study also attempted to control for the presence of particles by using glass beads and titanium. Unfortunately, as shown in Table 1, there is considerable variation in size and surface area. Figure 2 shows little effect of talc on IOSE cell viability (in contrast to the report by Buz'Zard). The vast majority of this paper focuses on gene expression changes in primarily mesothelial cells after exposure to these agents (figure 3, 4, 5, 6 and table 2, 3). As such (for the reasons just explained), the relevance of these experiments and the results is questionable. The only results directly relevant to ovarian cancer are shown in Table 4. A small number of genes in IOSE cells (2 at 8 hours with high concentrations) showed increased expression with asbestos with only 15 genes at 24 hours. More importantly, nonfibrous talc, titanium and glass beads showed no effect on IOSE cells.

C. Hamilton

In contrast to the above studies, there are more direct examinations of the effects of talc in *in vivo* models that undermine plaintiffs' experts' arguments. These types of experiments utilize rodent models, which can provide important detailed data on the relevance of talc exposure to the development of ovarian cancer. The first study was by Hamilton et al. entitled ***Effects of talc on the rat ovary***,[40] which exposed rat ovaries to high concentrations of talc by

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[38] Shukla et al., *Alterations in gene expression in human mesothelial cells correlate with mineral pathogenicity*. Am J Respir Cell Mol Biol. (July 2009) 41(1):114-23.

[39] Plunkett Rep. at 50. Dr. Clarke-Pearson opines that this study demonstrated *in vitro* that crocidolite asbestos and non-fibrous talc caused expression of genes in ovarian epithelial cells producing inflammatory cytokines. Clarke-Pearson Rep. at 4.

[40] Hamilton et al. (1984).

direct intrabursal injections. The animals were followed for up to 18 months and then had their tissues carefully examined after being sacrificed. While the animals showed cystic changes to their ovaries, there were no cases of ovarian cancer. Microscopic evaluation showed mostly unaffected surface epithelial cells, and, in four cases, papillary changes. These structures were composed of normal looking cells without any atypia. In addition, and perhaps more importantly, in five cases there were cortical foreign-body granulomas without any evidence of inflammatory infiltration found. There was no correlation between these granulomas and the papillary structures. These benign findings led the authors to consider alternative hypotheses, such as elevated hormone exposure.

The second study utilized an inhalation model for talc effects by exposing rats and mice to high concentrations of aerosolized talc.[41] This project, conducted by the National Toxicology Program, demonstrated that while there were lung toxicities induced by talc, there was a much more modest tumor effect. The few carcinomas in female rats could only be found in those with prolonged exposure to high levels of talc, and there was no greater incidence of malignant ovarian tumors in the exposed group.

D. Inflammation Theory

Inflammation has frequently been cited by plaintiffs' experts as the mechanism by which talc could increase the risk for ovarian cancer. In supposed support of this theory, they cite epidemiologic studies relating to the association of other inflammatory states with the increased risk of ovarian cancer and the use of NSAIDs and/or aspirin with an inverse risk of ovarian cancer. However, a close look at this data demonstrates that these studies do not support a relationship between inflammation induced by talc and the development of ovarian cancer.

Studies regarding inflammatory conditions and the risk of ovarian cancer have considered pelvic inflammatory disease (PID) and endometriosis.

1.) PID is an infection of the tubes and ovaries usually from a sexually transmitted disease. It is usually acute in nature but can become chronic. There have been multiple studies of the incidence of ovarian cancer in women with PID with inconsistent results.[42] A more recent larger nationwide cohort study in Taiwan demonstrated an association of PID with ovarian cancer.[43] This study had only a three-year follow-up, and as such, many of the cancers may have been present at the

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[41] National Toxicology Program, *Toxicology and Carcinogenesis Studies of Talc (CAS NO. 14807-96-6) in F344/N Raths and B6C3F$_1$ Mice (Inhalation Studies)*, Technical Report No. 421 (Sept. 1993).

[42] Rasmussen et al., *Pelvic Inflammatory Disease and the Risk of Ovarian Cancer and Borderline Ovarian Tumors: A Pooled Analysis of 13 Case-Control Studies*. Am J Epidemiol. (2017) 185(1): 8–20; Shen et al., *Risk of uterine, ovarian and breast cancer following pelvic inflammatory disease: a nationwide population-based retrospective cohort study*. BMC Cancer. (2016) 16(1):839; Zhou et al., *Pelvic inflammatory disease and the risk of ovarian cancer: a meta-analysis*. Cancer Causes Control. (2017) 28(5):415-428.

[43] Lin et al., *Risk of ovarian cancer in women with pelvic inflammatory disease: a population-based study*. Lancet Oncol. (2011) 12(9):900-4.

same time as PID. This is a serious flaw because it begs the question: Did PID increase the risk of ovarian cancer or did the cancer increase the risk of PID?

2.) Endometriosis is commonly cited as another inflammatory condition that is associated with ovarian cancer. This is also highly misleading, for several reasons. First, endometriosis is not associated with an increased risk of high grade serous ovarian cancer (HGSOC). HGSOC is the most common histology of ovarian cancer, has the highest mortality rate, and has been the primary focus of several of plaintiffs' experts. Endometriosis is associated with endometrioid and clear cell ovarian cancer histologies. Second, although there is frequently tissue reaction around endometriosis, including the infiltration of components of the immune system, endometriosis is really an ectopic displacement of endometrial tissue. This means that the endometrium is located outside of the uterus and as the endometrial tissue cycles, just like "normal" endometrial tissue, it will bleed and slough. This process obviously will lead to some local tissue reaction, and in some cases, fibrosis. There are no data that the tissue reaction is in fact the contributing factor versus the displaced endometrial cells. There are other well-known examples of displaced epithelial tissue increasing the risk of developing cancer. A good example is Barrett's esophagus, where gastric mucosa extends up into the distal esophagus where it does not usually reside, but in that position increases the risk of esophageal cancer.

The association between use of NSAIDs and/or aspirin and a decreased risk of ovarian cancer has also been cited as evidence for an inflammatory basis for the origin of ovarian cancer. But the supporting data do not make a persuasive argument. A meta-analysis of multiple epidemiologic studies examining the association of NSAIDs/aspirin use with ovarian cancer risk did not show any preventive effect.[44] This is an important analysis, as it is a very large study and includes all relevant studies up to 2012, concluding that there is no statistically significant association between NSAID use and the prevention of ovarian cancer. After extensive sorting, 21 studies met the appropriate criteria, and of these, 14 were case-control studies and 7 were cohort studies. The meta-analysis of these studies with regard to aspirin and NSAIDs showed no protective effects of these agents against the development of ovarian cancer. Even the subset analysis fails to show any meaningful association between the use of these drugs and a decreased risk of developing ovarian cancer. The authors conclude that, "[b]ased on this meta-analysis, the association between aspirin and non-aspirin NSAID use and ovarian cancer risk is weak."[45]

Some studies have evaluated markers of inflammation for possible correlations with ovarian cancer and these studies have been inconclusive or, if anything, have suggested that inflammation is not linked to ovarian cancer carcinogenesis. In one study by Trabert and others,

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[44] Baandrup et al., *Nonsteroidal anti-inflammatory drugs and risk of ovarian cancer: systematic review and meta-analysis of observational studies*. Acta Obstet Gynecol Scand. (2013) 92(3):245-55; Bonovas et al., *Do Nonsteroidal Anti-Inflammatory Drugs Affect the Risk of Developing Ovarian Cancer? A Meta-Analysis*, Brit. J. Clinical Pharmacology (2005) 60(2): 194-203; Ni et al., *Meta-Analysis on the Association Between Non-Steroidal Anti-Inflammatory Drug Use and Ovarian Cancer*, Brit. J. Clinical Pharmacology (2012) 75(1) 26-35.

[45] *Id.*

the authors investigated 46 inflammatory serum markers for possible association with ovarian cancer.[46] Of the 46 markers studied, only two – C-reactive protein (CrP) and Interleukin (IL-)-1α – were associated with the risk of developing ovarian cancer. The implications of this finding are unclear. As the authors acknowledged, because the markers were present in serum, the measurements can only report systemic levels, and thus "may not reflect levels in local sites of inflammation relevant to ovarian carcinogenesis, which may include the fallopian tubes, ovary, or endometriotic lesions." As a result, the implications of the findings are unclear and do not substantiate a link between local inflammation and ovarian cancer.

Another study identified three cohorts of women, including: (1) 60 women who had undergone risk-reducing removal of their ovaries and fallopian tubes because of hereditary risks for ovarian cancer; (2) 18 women who had undergone surgery for ovarian cancer without any known hereditary risks for the disease; and (3) 23 women (control group) who had undergone surgery to remove their fallopian tubes with benign diagnoses. The authors examined histological slides for markers of inflammation, including a lower ratio of ciliated cells, higher numbers of lymphocytes, and longer fimbria length (the fimbriae are tissues between the fallopian tubes and ovaries). Although the authors found increased inflammation between the cancer group and the control group, the trend was not statistically significant. The authors also noted that age could have been a confounder because the mean age of women with cancer was four years higher than the women in the control group. The authors concluded that "no significant correlation was made between serous carcinoma and histological signs of inflammation" and that more research is necessary "to further evaluate the role of inflammation in carcinogenesis in the fallopian tube."[47]

IV. DR. SAED'S PLAINTIFF-FUNDED RESEARCH

One of plaintiffs' experts, Dr. Ghassan Saed, opines that his work has established a plausible biological mechanism by which ovarian cells exposed to talc could develop cancer, an opinion he sets forth both in a report prepared for this litigation and in an in-press manuscript.

As discussed at length below, Dr. Saed's research suffers from a number of severe methodological flaws, rendering it at times uninterpretable and certainly unreliable. None of the findings he reports has been shown to cause cancer in general, much less ovarian cancer. Dr. Saed also makes some of the same unsubstantiated assumptions as plaintiffs' other experts about the significance of alterations in gene expression or the effects of inflammation or levels of ROS. Moreover, he has not made any effort to replicate his *in vitro* findings *in vivo*, as noted by one reviewer for *Gynecologic Oncology*, which rejected his manuscript.[48] Because cells in living tissue react differently than cells in a laboratory, it is widely accepted that, while *in vitro* studies

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[46] Trabert et al., *Pre-diagnostic serum levels of inflammation markers and risk of ovarian cancer in the Prostate, Lung, Colorectal and Ovarian Cancer (PLCO) Screening Trial*. Gyn. Onc. (2014) 135:297-304.

[47] Malmberg et al., *Serous tubal intraepithelial carcinoma, chronic fallopian tube injury, and serous carcinoma development*. Virchows Arch. (2016) 468(6):707-13.

[48] Saed II Dep. Ex. 35 (Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision).

provide a valuable starting point, no conclusions about human health can be drawn from them. As another *Gynecologic Oncology* reviewer explained, "the present data are insufficient to support the claim that talcum is central to the development of ovarian cancer."[49]

Due to the flaws in Dr. Saed's methodology, his work cannot support even his more modest conclusions. His opinion is subject to many of the same problems identified above in other work. For example, Dr. Saed presumes that perineal talc use would result in exposure of ovarian tissue to talc through migration, but such migration has not been established. In addition, Dr. Saed's methodology is flawed in a number of different ways. Most importantly, he did not use rigorous methods in his research, and his report is filled with speculation and guessing that he mischaracterizes as scientific knowledge.

A. Dr. Saed's Report

Dr. Saed's report is filled with generalities regarding ovarian cancer that are not supported by any citations and reflect a superficial understanding of the disease. For example:

- He refers to "malignant overgrowth versus a benign overgrowth, specifically postoperative adhesions."[50] But he does not explain what postoperative adhesions have to do with ovarian cancer development, and from a scientific standpoint, there is no generally accepted connection.

- On pages 5-6, he states that "two enzymes, MPO and iNOS, work together to inhibit apoptosis, a hallmark of ovarian cancer." There are no references for this statement. I know of no scientific basis for stating that these two enzymes inhibit apoptosis or that apoptosis is critical for the development or progress of ovarian cancer. Notably, there is no reference supporting that apoptosis is a "hallmark" of ovarian cancer.

- There is an extensive discussion on page 7 concerning CA-125 and HE4 as serum biomarkers for ovarian cancer. It is completely unclear what this has to do with the role of talc in ovarian cancer development. The fact that CA-125 can be elevated in cases of ovarian cancer does not mean it can contribute to cancer causation. To take a simple example, a fever may be a "biomarker" for a bacterial or viral infection, but the fever obviously does not contribute to causing the infection. While HE4 has recently been proposed as a possible ovarian cancer biomarker as well in certain studies, the suggestion that it can contribute to cancer causation is even more speculative.

- On page 17, Dr. Saed makes the statement: "Consistent with these findings, recent studies from my laboratory have shown that talc enhances cell proliferation and induces an inhibition in apoptosis in EOC cells, but more importantly in normal cells, suggesting talc is a stimulus to the development of the oncogenic

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49 *Id*.

50 Saed Rep. at 2.

phenotype." As discussed below, he includes no reference for this statement. But even if it is true, cell proliferation and inhibited apoptosis can also occur in healthy and non-malignant cells and therefore cannot be broadly characterized as an "oncogenic phenotype." I note with concern that Dr. Saed does not seem to appreciate this very basic, well-established fact. At his deposition, Dr. Saed expressed belief that his tests showed development of neoplastic cells because he reported finding "[p]roliferation," which he claimed was "an indirect measure of the beginning of a transformation."[51] This is not so. One might well expect to see accelerated cell proliferation where a neoplastic transformation has occurred, but accelerated cell proliferation itself does not serve to identify the existence of a neoplastic transformation.[52] Many normal tissues proliferate, including the endometrium, cervical epithelium, colonic epithelium and even skin.

- On page 5, he discusses the GSH/GSSG complex and its stimulation of the activity of GS-X-MRP1 efflux pump and its relation to the development of resistance to chemotherapeutic drugs. This is obviously irrelevant to the development of ovarian cancer, and GS-X-MRP1 efflux pump activity has no clinical significance in the treatment of ovarian cancer either.

Dr. Saed's experiments do not reflect an in-depth understanding of ovarian cancer and are methodologically flawed in a number of ways. Most notably:

- Dr. Saed's cell line work involves cancer cell lines, which are not high grade serous ovarian cancer (HGSOC) cell lines. TOV112D (endometrioid), SKOV-3 (endometrioid or clear cell), and A2780 are not HGSOC cell lines, and as such have no relevance to HGSOC, the most common subtype of ovarian cancer and a key focus of the epidemiology and plaintiffs' expert. Therefore, these cell line models and the data derived from them do not support the role of talc in the development of ovarian cancer. This is a serious flaw in Dr. Saed's work, and it reflects a certain lack of understanding of the state of the science in ovarian cancer research. Similarly, only one of his three normal cell lines, FT33, was from fallopian cells – the relevant cell type given that most ovarian cancer is now known to start in the fallopian tubes. And this cell line was immortalized, meaning that, by definition, the cells were not normal and the impact of talc on normal fallopian cells remains untested.

- Most of the experiments are conducted using doses (5-100 ug/ml) applied directly to ovarian cells that are inconsistent with exposure of ovarian tissue to talc that women might experience dusting their perineum with powder (indeed, there likely is no such exposure, as discussed above). These are enormous doses and there is no data to support that the doses used in these experiments are what the female genital tract would be exposed to from the dusting of the perineum, even

[51] Saed II Dep. 464:2-11.

[52] Chaffer & Weinberg, *How does multistep tumorigenesis really proceed?* Cancer Discov. (2015) 5(1):22-4.

> assuming that some talc could migrate up to the fallopian tubes or ovaries. It is certainly not consistent with the reported detection of talc particles in ovarian cancers, which are exceedingly small in numbers.

There is considerable discussion about SNPs in the document. It is exceedingly difficult to follow and understand. There are multiple references to mutations in SNPs, but it is unclear what the author is referring to. SNPs are inherited and exist at birth and therefore each person has a unique pattern of SNPs. It might be that Dr. Saed is referring to this fact, or he could be hypothesizing somatic mutations within the tumor or even the fertilized egg (or perhaps something else entirely). If Dr. Saed is claiming that talc treatment of the cells caused the development of specific SNPs that did not previously exist through mutation, he has provided no evidence of that hypothesized effect or how it would work. The imprecision in these statements is important, as it reflects shortfalls in the thought process and expertise that went into the report.

There is also an extensive discussion about the impact of SNPs on protein function and their association with ovarian cancer. Dr. Saed makes the argument that many of these SNPs result in amino acid changes and the resultant proteins have altered functions. This results in changes in oxidants and antioxidants, which, it is argued, affects ovarian cancer risk and development. The data presented are very poor and confusing. None of the studies on which Dr. Saed relies show any relationship between the SNPs he identified and increased risk of ovarian cancer. Most of what is referenced are general reviews or papers discussing SNPs in "oxidative DNA repair genes and redox genes with human cancer susceptibility."[53] The specific relevance of this for ovarian cancer is unclear. Further SNP data is extracted from ovarian cancer Genome Wide Association Studies (GWAS) studies, and by and large they demonstrate small but statistically significant associations of specific SNPs with the risk for ovarian cancer. These SNPs have had no clinical impact on the management of ovarian cancer patients or cancer prevention/screening. In addition, the mechanism(s) by which these SNPs affect cell behavior remains completely unknown; in particular, it is unknown whether any of these SNPs affect the function of the proteins synthesized from the affected DNA, much less whether the SNPs can ultimately affect the redox state of cells.

Dr. Saed also utilizes a mixture of other investigators' work in systems other than ovarian cancer, SNP data in ovarian cancer patients and his own work using ovarian cancer cell lines (most of which are not HGSOC) to support the contention that these genes and their SNPs play a role in the development of ovarian cancer. The former work, which, as mentioned, does not involve ovarian or fallopian tissue, is irrelevant to ovarian cancer, as complexity of tissues and their biochemistry can make functional results completely different from one human tissue to another.

Dr. Saed's interpretation of his own work is even more problematic. With respect to the ovarian cancer SNP data, he refers to the presence of a particular SNP with patient survival. But he does not explain what a prognostic marker has to do with risk markers. Is he arguing they are the same? As discussed in the context of proteins, prognostic markers may be associated with ovarian cancer or with ovarian cancer survival without playing a role in causation. On the bottom

[53] Saed Rep. at 7.

of page 8, Dr. Saed lists many SNPs associated with the risk of ovarian cancer (***notably, none of which was found in his experiment***) and admits that they are "near" genes, which are not associated with redox/oxidative stress. In fact, this is correct; the SNPs are just genomic markers and may identify neighboring genes, which are the actual ones that ascribe the risk. The genes in which the SNP mutation actually occurred are likely to have no role in oxidative stress or redox.

Dr. Saed's research is also unreliable because it is impossible to understand exactly what he did. There is little or no primary data, and what is described is insufficient. In short, he offers a host of conclusions about the results of his study, but no data that would allow those conclusions to be evaluated or replicated.

- On page 18, it is stated that "treatment of normal or ovarian cancer cells with talc resulted in a significant increase in MPO and iNOS." There is no reference or data presented. Although I understand that Dr. Saed has produced lab books that are supposed to contain the underlying data, those data have proven unreliable in several respects. Specifically, at his deposition, Dr. Saed repeatedly admitted to what he called "typos" or errors concerning fundamental issues of dose, statistical significance and time of treatment.[54] He similarly acknowledged errors that he said he "can't explain" in calculations contained within his lab books – the raw data on which his report and opinions are based.[55] The pervasiveness of these errors virtually ensures that there are other errors pertaining to critical points that reviewers of Dr. Saed's work are in no position to uncover, calling all of his work and conclusions into even more doubt and rendering them scientifically unreliable.

- Dr. Saed uses cell lines (TOV112D, SKOV-3 and A2780) in his reported experiments, which do not reflect the epidemiologic data relating talc to the risk of ovarian cancer, and more importantly, the SNP data. As discussed above, none of his cell lines were HGSOC cell lines, and both the epidemiology and the SNP data studied primarily HGSOC cases.

- Dr. Saed also states that, "[c]onsistent with this finding, it has previously been reported that acquisition of chemoresistance by ovarian cancer cells is associated with a switch from the *GPX1* SNP genotype to the normal *GPX1* genotype." Dr. Saed does not opine that talc makes existing ovarian cancer more difficult to treat, but rather that it causes ovarian cancer to develop in the first place. What does chemoresistance have to do with ovarian cancer development?

- Additionally, Dr. Saed reports: "our results showed that talc treatment was associated with a genotype switch from common C/C genotype in *NOS2* in untreated cells to T/T, the SNP genotype, in talc treated cells, except in A2780 and TOV112D."[56] What is a genotypic switch? Is he reporting a direct mutation in

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54 Saed II Dep. 403:3-407:11, 416:9-417:7, 457:21-458:25, 542:3-15.

55 *Id.* 450:24-453:24.

56 Saed Rep. at 19.

the DNA? There is no suggestion in the literature that talc interacts with DNA and no data in his report or lab books providing an explanation of how that would be possible.

- Dr. Saed argues in his report that exposure to chemotherapy alters SNP profiles and this is important for the development of resistance. As mentioned above, Dr. Saed purports to offer an opinion about the development of ovarian cancer, not about its susceptibility to treatment. It is entirely unclear what chemoresistant SNP profiles have to do with talc and the development of ovarian cancer. Since 80% of ovarian cancers are drug-sensitive at diagnosis, if talc is associated with the development of resistance, then it is not associated with cancer development. Chemoresistant ovarian cancer develops after multiple recurrences and years of exposure to different drug regimens. What Dr. Saed describes is poorly presented, and more importantly makes little scientific sense.

B. Dr. Saed's Recent Manuscript

The manuscript entitled *Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer* by Fletcher et al.[57] (for which Dr. Saed is the corresponding author) describes a series of experiments attempting to demonstrate evidence for the role of talc in the development of ovarian cancer. Dr. Saed acknowledged at his deposition that this article was based on the same research as his expert report. Unsurprisingly, this manuscript has serious methodologic, experimental, and analysis flaws, including many of the same ones already identified. Specifically:

1. As was the case in his report, none of the cancer cell lines used are HGSOC cell lines. SKOV-3, A2780, and TOV112D are not of serous origin. It would be the same if the authors were using lung or colon cancer cell lines. These experiments are not relevant to the suggested role of talc in HGSOC. Similarly, just one of the normal cell lines is of fallopian origin, and one of them is not even derived from female reproductive tissue at all, but rather from macrophages.

2. Figure 1 shows a decrease in CAT mRNA expression only at high concentrations of talc. There is no evidence that this is the concentration of talc that women are exposed to when dusting their perineum. Talc does not show a decrease in SOD3 except at the 100ug/ml concentration. The control – a solution of pure DMSO – shows an 80% decrease by itself. The effect of the control suggests that to the extent Fletcher and Saed found that extremely high talc concentrations altered mRNA expression, the effect results from cell culture conditions, and not talc. Additionally, the mRNA levels were normalized to B-actin. Is this appropriate, as opposed to other house-keeping genes? Saed and Fletcher make no effort to justify their selection of B-actin, and the reported changes of gene expression levels could actually reflect different stabilities of the target genes versus

[57] Saed I Dep. Ex. 8 (Fletcher NM, Harper AK, Memaj I, Fan R, Morris RT, Saed GM, *Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer* (2019) (unpublished manuscript)).

coupled with instable levels of the control B-actin. The manuscript does not address this possibility, which goes to the reliability of the reported results.

3. It is unclear whether figure 1 shows enzyme activity or protein levels. It is labeled ELISA (a test used to detect protein levels) and the text reports protein levels. Yet, the ordinate reflects some sort of enzyme activity. There are no methods describing the procedure used to generate the results, making them impossible to interpret.

4. The CA125 experiments showing increased CA125 levels under talc treatment have no relevance to the development of ovarian cancer. CA125 has no proven role in the development of ovarian cancer. As mentioned above, CA125 is sometimes used as an ovarian cancer biomarker, but that does not mean it can contribute to causing ovarian cancer. Moreover, CA125 gene regulation is controlled by many transcription factors.

5. Figure 5 utilizes the MTT assay as a measure for cellular proliferation. A direct cell count is necessary to ensure that the MTT result is correct. The MTT assay is a colorimetric assay that measures metabolic activity, which can be a proxy for a cell count. But MTT results could be misleading. As a matter of basic science, the MTT assay can only indirectly measure cell proliferation because what it detects is enzymatic activity, which is supposed to correlate to cell counts, but it has long been understood that this indirect measurement is subject to potential interference that can significantly impair its accuracy. In one study examining the accuracy of an MTT assay in control cells to which DMSO had been applied, for example, the true number of control cells was found to be "10-fold higher" than reported by the MTT assay.[58] Dr. Saed is apparently unaware of this problem.

6. Figure 6 reports decreased apoptosis in talc treated cells. There is no description of other mechanisms of cell death, including necrosis, etc. Because the study does not report other mechanisms of cell death, it is possible that overall cell death rates are the same in treated and non-treated cells. If the controls are, in fact, dying faster than the talc treated cells, then is there talc-induced proliferation?

7. The SNP data are very difficult to understand. Indeed, one of the reviewers for *Gynecologic Oncology* noted that "[t]he significance of SNP alterations should be further clarified."[59] To the extent I can understand the data, they seem to suggest that treatment of the cultures (with decreased cellular apoptosis) somehow undergoes a specific DNA-based switch. It is not clear to me what a switch means. Do the authors mean a mutation of one of the alleles or selection of one of the alleles? The manuscript provides no data to explain why either of these things would happen. As mentioned, there are no data I am aware of – either in the literature or in Fletcher and Saed's results – that a particle such as talc could specifically mutate DNA, and it is not apparent how it could. How does talc

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[58] Plumb et al., *Effects of the pH dependence of 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide-formazan absorption on chemosensitivity determined by a novel tetrazolium-based assay*. Cancer Res. (1989) 49:4435-4440.

[59] Saed II Dep. Ex. 35 (Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision).

enter the cell? How does it get into the nucleus? If it does not do those things, what is the mechanism by which it can alter DNA? A secondary process resulting for talc would not be expected to be specific for the DNA sequence. If selection, rather than mutation, is involved, then, again, what is the mechanism of selection? There are no data to address any of these questions, and quite frankly, the results as described are not plausible.

V. CONCLUSION

Much remains to be understood about ovarian carcinogenesis, and unfortunately, nothing plaintiffs' experts offer in their reports or depositions advances our understanding. Talc is not generally accepted as a cause of ovarian cancer. In fact, much of what plaintiffs' experts cite as supposed support for their conclusions on biological plausibility reflects work that attempted to – but did not – clarify a potential causal relationship between talc and ovarian cancer (and in many cases added significant support to the null hypothesis – i.e., that talc *does not* cause ovarian cancer). The little new science that plaintiffs' experts attempted to contribute to this inquiry – principally in the form of Dr. Saed's experiments – is deeply flawed at many levels as described in this report and, even ignoring these flaws, does not bring us any closer to establishing any hypothesized causal connection between talc use and ovarian cancer.

Materials Reviewed and Considered by Dr. Michael Birrer

1. Baandrup et al., Nonsteroidal anti-inflammatory drugs and risk of ovarian cancer: systematic review and meta-analysis of observational studies. Acta Obstet Gynecol Scand. (2013) 92(3):245-55.

2. Berge et al., *Genital use of talc and risk of ovarian cancer: a meta-analysis*. Eu J of Cancer Prev. (2018) 27:248-257.

3. Bonovas et al., *Do Nonsteroidal Anti-Inflammatory Drugs Affect the Risk of Developing Ovarian Cancer? A Meta-Analysis,*, Brit. J. Clinical Pharmacology (2005) 60(2): 194-203.

4. Boorman et al., The Lack of an Ovarian Effect of Lifetime Talc Exposure in F344/N Rats and B6C3F1 Mice. Regul Toxicol Pharmacol. (1995) 21(2):242-3.

5. Booth et al., Risk factors for ovarian cancer: a case-control study. Br J Cancer (1989) 60:592-8.

6. Buz'Zard & Lau, Pycnogenol reduces talc-induced neoplastic transformation in human ovarian cell cultures. Phytother Res. (2007) 21(6):579-86.

7. Camargo et al., Occupational Exposure to Asbestos and Ovarian Cancer: A Meta-analysis. Environ Health Perspect (2011) 119(9):1211–1217.

8. Cancer Genome Atlas Research Network, Integrated genomic analyses of ovarian carcinoma. Nature (2011) 474(7353):609-15.

9. Cannistra, Cancer of the ovary. N Engl J Med. (2004) 351(24):2519-29.

10. Carr, Talc: Consumer Uses and Health Perspectives. Reg Toxicol Pharmacol (1995) 21:211-215.

11. Chaffer & Weinberg, How does multistep tumorigenesis really proceed? Cancer Discov. (2015) 5(1):22-4.

12. Chang & Risch, Perineal talc exposure and risk of ovarian carcinoma. Cancer (1997) 79:2396-401.

13. Chen et al., Risk factors for epithelial ovarian cancer in Beijing, China. Int J Epidemiol (1992) 21:23-9.

14. Cho & Shih, Ovarian cancer. Annu Rev Pathol. (2009) 4:287-313.

15. Cook et al., Perineal powder exposure and the risk of ovarian cancer. Am J Epidemiol (1997) 145:459-65.

16. Cramer & Xu, Epidemiologic evidence for uterine growth factors in the pathogenesis of ovarian cancer. Ann Epidemiol (1995) 5:310-4.

17. Cramer et al., Genital talc exposure and risk of ovarian cancer. (1999) 81(3) Int J Cancer. 351.

18. Cramer et al., Ovarian cancer and talc: a case-control study. Cancer (1982) 50:372-6.

19. Cramer et al., Presence of talc in pelvic lymph nodes of a woman with ovarian cancer and long-term genital exposure to cosmetic talc. Obstet Gynecol. (2007) 110(2 Pt 2):498-501.

20. Cramer et al., The Association Between Talc Use and Ovarian Cancer: A Retrospective Case control study in two US States. Epidemiol. (2016) 27(3):334-346.

21. Cramer, Perineal talc exposure and subsequent epithelial ovarian cancer: a case-control study. Obstet Gynecol (1999) 94:160-1.

22. Crawford et al., Perineal powder use and risk of endometrial cancer in postmenopausal women. Cancer Causes Control 2012; 23:1673-80.

23. Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738)

24. Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738)

25. Driscoll et al., Effects of particle exposure and particle-elicited inflammatory cells on mutation in rat alveolar epithelial cells. Carcinogenesis. (1997) 18(2):423-30.

26. DS Biotech, LLC Invoice to Beasley Allen, from Ghassan Saed, Ph.D., dated February 8, 2019 (Ex. 22 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

27. Egli & Newton, The transport of carbon particles in the human female *reproductive tract.* Fertil Steril. (1961) 12:151-5.

28. Endo-Capron et al., Some in vivo and in vitro studies carried out with talc samples, NATO ASI Series G, 1990.

29. Expert Report of Anne McTiernan, M.D., Ph.D., Nov. 16, 2018 (MDL No. 2738)

30. Expert Report of Arch Carson, M.D., Ph.D., Nov. 16, 2018 (MDL No. 2738)

31. Expert Report of Daniel L. Clarke-Pearson, M.D., Nov. 16, 2018 (MDL No. 2738)

32. Expert Report of David A. Kessler, M.D., Nov. 16, 2018 (MDL No. 2738)

33. Expert Report of Ellen Blair Smith, M.D., Nov. 16, 2018 (MDL No. 2738)

34. Expert Report of Ghassan M. Saed, Ph.D., Nov. 16, 2018 (MDL No. 2738)

35. Expert Report of Jack Siemiatycki, M.Sc., Ph.D., Nov. 16, 2018 (MDL No. 2738)

36. Expert Report of Judith Wolf, M.D., Nov. 16, 2018 (MDL No. 2738)

37. Expert Report of Judith Zelikoff, Ph.D., Nov. 16, 2018 (MDL No. 2738)

38. Expert Report of Laura M. Plunkett, Ph.D., DABT, Nov. 16, 2018 (MDL No. 2738)

39. Expert Report of Patricia Moorman, M.S.P.H., Ph.D., Nov. 16, 2018 (MDL No. 2738)

40. Expert Report of Rebecca Smith-Bindman, M.D., Nov. 15, 2018 (MDL No. 2738)

41. Expert Report of Sarah E. Kane, M.D., Nov. 15, 2018 (MDL No. 2738)

42. Expert Report of Shawn Levy, Ph.D., Nov. 16, 2018 (MDL No. 2738)

43. Expert Report of Sonal Singh, M.D., M.P.H., Nov. 16, 2018 (MDL No. 2738)

44. Faber et al., Oral contraceptive use and impact of cumulative intake of estrogen and progestin on risk of ovarian cancer. Cancer Causes Control. (2013) 24(12):2197-2206.

45. Ferrer et al., Influence of particle size on extrapleural talc dissemination after talc slurry pleurodesis. Chest. (2002) 122(3):1018-27.

46. Fiume et al., Safety Assessment of Talc as Used in Cosmetics. Int J Toxicol. (2015) 34(1 Suppl):66S-129S.

47. Fletcher NM, Harper AK, Memaj I, Fan R, Morris RT, Saed GM, Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer (Oct. 10, 2018) (unpublished manuscript) (Ex. 38 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

48. Fletcher NM, Harper AK, Memaj I, Fan R, Morris RT, Saed GM, Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer (Jan. 3, 2019) (unpublished manuscript) (Exs. 7 & 8 to the Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))

49. Food and Drug Administration, Letter from Musser SM to Epstein SS Re: Docket Numbers 94P-0420 and FDA-2008-P-0309-0001/CP. Date stamped: April 1, 2014. April 1, 2014 FDA Denial of 1994 and 2008 Petitions.

50. Food and Drug Administration, Statement on Talc (2015).

51. Gates et al., Risk Factors for Epithelial Ovarian Cancer by Histologic Subtype. Am J Epidemiol (2010) 171:45-53.

52. Gates et al., Talc use, variants of the GSTM1, GSTT1, and NAT2 genes, and risk of epithelial ovarian cancer. Cancer Epidemiol Biomarkers Prev. (2008) 17:2436-44.

53. Genofre et al., Talc pleurodesis: evidence of systemic inflammatory response to small size talc particles. Respir Med. (2009) 103(1):91-7.

54. Gertig et al., Prospective study of talc use and ovarian cancer. J Natl Cancer Inst. (2000) 92(3):249-52.

55. Ghassan Saed's PCR EOC SRI Notebook (Ex. 9 to the Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))

56. Ghassan Saed's Talc Study Lab Notebook – Preliminary Study (Ex. 23 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

57. Godard et al., Risk factors for familial and sporadic ovarian cancer among French Canadians: a case-control study. Am J Obstet Gynecol (1998) 179:403-10.

58. Gonzalez, Douching, Talc Use, and Risk of Ovarian Cancer. Epidemiol. (2016) 27(6):797-802.

59. Green et al., Tubal sterilization, hysterectomy and decreased risk of ovarian cancer. Survey of Women's Health Study Group. Int J Cancer (1997) 71:948-51.

60. Gross et al., A Meta-Analytical Approach Examining the Potential Relationship Between Talc Exposure and Ovarian Cancer. J Exp Anal Eviron Epidemiol (1995) 5:181-195.

61. Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision (Ex. 35 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

62. Hamilton et al., Effects of Talc on the Rat Ovary. Br J Exp Pathol. (1984) 65(1):101-6.

63. Harlow & Hartge, A review of perineal talc exposure and risk of ovarian cancer. Regul Toxicol Pharmacol (1995) 21:254-60.

64. Harlow & Weiss, A case-control study of borderline ovarian tumors: the influence of perineal exposure to talc. Am J Epidemiol (1989) 130:390-4.

65. Harlow et al., Perineal exposure to talc and ovarian cancer risk. Obstet Gynecol (1992) 80:19-26.

66. Hartge et al., Talc and ovarian cancer. JAMA (1983) 250:1844.

67. Heller et al., Asbestos exposure and ovarian fiber burden. Am. J. Indus. Med. (1996) 29:435-439.

68. Heller et al., Correlation of asbestos fiber burdens in fallopian tubes and ovarian tissue. Gynecol. (1999) 181:346-47.

69. Heller et al., Presence of asbestos in peritoneal malignant mesotheliomas in women. Int. J. Gynecol. Cancer. (1999) 9:452-455.

70. Heller et al., The relationship between perineal cosmetic talc usage and ovarian talc particle burden. Am J Obstet Gynecol. (1996) 174(5):1507-10.

71. Henderson et al., Talc and carcinoma of the ovary and cervix. J Obstet Gynaecol Br Commonw. (1971) 78(3):266-72.

72. Henderson et al., Talc in normal and malignant ovarian tissue. Lancet. (1979) 1(8114):499.

73. Hillegass et al., Utilization of Gene Profiling and Proteomics to Determine Mineral Pathogenicity in a Human Mesothelial Cell Line (LP9/TERT-1). Journal of Toxicology and Environmental Health, Part A, 73::423-436-2010

74. Hollinger, Pulmonary toxicity of inhaled and intravenous talc. Toxicol Lett. (1990) 52(2):121-7.

75. Houghton et al., Perineal Powder Use and Risk of Ovarian Cancer. J Natl Cancer Inst (2014) 106(90); dju208.

76. Huncharek et al., Perineal Application of Cosmetic Talc and Risk of Invasive Epithelial Ovarian Cancer: A Meta-analysis of 11,933 Subjects from 16 Observational Studies. Anticancer Research (2003) 23:1955-1960.

77. IARC, Arsenic Metals, Fibres, and Dusts, Vol 100 C.

78. IARC, Carbon Black, Titanium Dioxide, and Talc, Vol 93.

79. Jordan et al., Breastfeeding and risk of epithelial ovarian cancer. Cancer Causes Control. (2010) 21(1):109-116.

80. Jordan et al., Risk factors for benign serous and mucinous epithelial ovarian tumors. Obstet Gynecol (2007) 109:647-54.

81. Karageorgi et al., Perineal use of talcum powder and endometrial cancer risk. Cancer Epidemiol Biomarkers Prev (2010) 19:1269-1275.

82. Keskin et al., Does long-term talc exposure have a carcinogenic effect on the female genital system of rats? An experimental pilot study. Arch Gynecol Obstet (2009) 280:925-931.

83. Korsic et al., Talc pleurodesis improves survival of patients with malignant pleural effusions: case-control study Wien Klin Wochenschr DOI 10.1007/s00508-015-0716-8

84. Kotsopoulos et al., Ovarian cancer risk factors by tumor dominance, a surrogate for cell of origin. Int J Cancer (2013) 133:730-740.

85. Kreyling, Semmler-Behnke & Möller, Ultrafine particle-lung interaction: Does size matter? J Aerosol Med. (2006) 19(1):74-83.

86. Kunz et al., The uterine peristaltic pump. Normal and impeded sperm transport within the female genital tract. Adv Exp Med Biol. (1997) 424:267-77.

87. Kurman & Shih, The origin and pathogenesis of epithelial ovarian cancer: a proposed unifying theory. Am. J. Surg. Pathol. (2010). 34:433–443.

88. Lab Notebook, SAED000001(color)-SAED000097(color) (Ex. 1 to the Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))

89. Lacey et al., Menopausal Hormone Replacement Therapy and Risk of Ovarian Cancer. JAMA Network. (2002) 288(3):334-341.

90. Langseth et al., Perineal use of talc and risk of ovarian cancer. J Epidemiol Comm Health (2008) 62(4):358-360.

91. Letter to Lawrence Layman, Editor, Reproductive Sciences, from Ghassan Saed, Ph.D., dated Jan. 2, 2019 (Ex. 40 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

92. Lin et al., Risk of ovarian cancer in women with pelvic inflammatory disease: a population-based study. Lancet Oncol. (2011) 12(9):900-4.

93. Litton Bionetics, Inc., Summary of Mutagenicity Screening Studies Host-Mediated Assay Cytogenetics Dominant Lethal Assay, December 9, 1974.

94. Malmberg et al., Serous tubal intraepithelial carcinoma, chronic fallopian tube injury, and serous carcinoma development. Virchows Arch. (2016) 468(6):707-13.

95. Merritt et al., Talcum powder chronic pelvic inflammation and NSAIDS in relation to risk. Int J Cancer (2007) 122:170-176.

96. Mills et al., Perineal Talc Exposure and Epithelial Ovarian Cancer Risk in the Central Valley of California. (2004) 112 Int'l J. Cancer 458.

97. Muscat & Huncharek, Perineal talc use and ovarian cancer: a critical review. Eur J Cancer Prev (2008) 17:139-46.

98. Nakane, Translocation of particles deposited in the respiratory system: a systematic review and statistical analysis. Environ Health Prev Med. (2012) 17(4):263-74.

99. National Cancer Institute, Ovarian, Fallopian Tube, and Primary Peritoneal Cancer Prevention (PDQ), Health Professional Version, https://www.cancer.gov/types/ovarian/hp/ovarian-epithelial-treatment-pdq.

100. National Toxicology Program, Toxicology and Carcinogenesis Studies of Talc (CAS NO. 14807-96-6) in F344/N Raths and B6C3F1 Mice (Inhalation Studies), Technical Report No. 421 (Sept. 1993).

101. Neill et al., Use of talcum powder and endometrial cancer risk. Cancer Causes Control (2012) 23:513-519.

102. Ness et al., Factors related to inflammation of the ovarian epithelium and risk of ovarian cancer. Epidemiology (2000) 11:111-7.

103. Ness, Does talc exposure cause ovarian cancer? IGCS-0015 Ovarian Cancer. Int J Gynecol Cancer (2015) 25(Suppl 1):51.

104. Ni et al., Meta-Analysis on the Association Between Non-Steroidal Anti-Inflammatory Drug Use and Ovarian Cancer, Brit. J. Clinical Pharmacology (2012) 75(1) 26-35.

105. Penninkilampi & Eslick, Perineal Talc Use and Ovarian Cancer: A Systematic Review and Meta-Analysis. Epidemiology (2018) 29(1):41-49.

106. Peres et al., Racial/ethnic differences in the epidemiology of ovarian cancer: a pooled analysis of 12 case-control studies. Int J Epidemiol (2017) 1–13.

107. Peters et al., Translocation and potential neurological effects of fine and ultrafine particles a critical update. Part Fibre Toxicol. (2006) 8;3:13.

108. Plumb et al., Effects of the pH dependence of 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide-formazan absorption on chemosensitivity determined by a novel tetrazolium-based assay. Cancer Res. (1989) 49:4435-4440.

109. Purdie et al., Reproductive and other factors and risk of epithelial ovarian cancer: an Australian case-control study. Survey of Women's Health Study Group. Int J Cancer (1995) 62:678-84.

110. Rasmussen et al., Pelvic Inflammatory Disease and the Risk of Ovarian Cancer and Borderline Ovarian Tumors: A Pooled Analysis of 13 Case-Control Studies. Am J Epidemiol. (2017) 185(1): 8–20.

111. Reid et al., Does Exposure to Asbestos Cause Ovarian Cancer? A Systematic Literature Review and Meta-analysis. Cancer Epidemiol Biomarkers Prev. (2011) 20(7):1287-95.

112. Rosenblatt et al., Genital powder exposure and the risk of epithelial ovarian cancer. Cancer Causes Control (2011) 22:737-742.

113. Rosenblatt et al., Mineral fiber exposure and the development of ovarian cancer. Gynecol Oncol (1992) 45:20-5.

114. Rossi et al., Acute inflammatory response secondary to intrapleural administration of two types of talc. Eur Respir J. (2010) 35(2):396-401.

115. Schildkraut et al., Association between Body Powder Use and Ovarian Cancer: the African American Cancer Epidemiology Study (AACES). Cancer Epidemiol Biomarkers Prev. (2016) 25(10):1411-7.

116. Shen et al., Risk of uterine, ovarian and breast cancer following pelvic inflammatory disease: a nationwide population-based retrospective cohort study. BMC Cancer. (2016) 16(1):839.

117. Shukla et al., Alterations in gene expression in human mesothelial cells correlate with mineral pathogenicity. Am J Respir Cell Mol Biol. (July 2009) 41(1):114-23.

118. Sjösten, Ellis & Edelstam, Retrograde migration of glove powder in the human female genital tract. Hum Reprod. (2004) 19(4):991-5.

119. Terry et al., Genital powder use and risk of ovarian cancer: a pooled analysis of 8,525 cases and 9,859 controls. Cancer Prev Res (Phila). (2013) 6(8):811-21.

120. Tiourin et al., Tubal Ligation Induces Quiescence in the Epithelia of the Fallopian Tube Fimbria. Reprod Sci. (2015) 22(10):1262-71.

121. Trabert et al., Aspirin, Nonaspirin Nonsteroidal Anti-inflammatory Drug, and Acetaminophen Use and Risk of Invasive Epithelial Ovarian Cancer: A Pooled Analysis in the Ovarian Cancer Association Consortium. J Natl Cancer Inst. (2014) 106(2):djt431.

122. Trabert et al., Pre-diagnostic serum levels of inflammation markers and risk of ovarian cancer in the Prostate, Lung, Colorectal and Ovarian Cancer (PLCO) Screening Trial. Gyn. Onc. (2014) 135:297-304.

123. Tzonou et al., Hair dyes, analgesics, tranquilizers and perineal talc application as risk factors for ovarian cancer. Int J Cancer (1993) 55:408-10.

124. Venter & Iturralde, Migration of a particulate radioactive tracer from the vagina to the peritoneal cavity and ovaries. S Afr Med J. (1979) 55(23):917-9.

125. Viskum K, et al. Long term sequelae after talc pleurodesis for spontaneous pneumothorax. Pneumologie. (1989) 43:105-6.

126. Wentzensen et al., Talc Use and Ovarian Cancer: Epidemiology Between a Rock and a Hard Place. J Natl Cancer Inst. (2014) 106(9) DOI:10.1093/njci/dju260.

127. Werebe et al., Systemic distribution of talc after intrapleural administration in rats. Chest. (1999) 115(1):190-3.

128. Whittemore et al., Personal and environmental characteristics related to epithelial ovarian cancer. II. Exposures to talcum powder, tobacco, alcohol, and coffee. Am J Epidemiol (1988) 128:1228-40.

129. Wong et al., Perineal talc exposure and subsequent epithelial ovarian cancer: a case-control study. Obstet Gynecol (1999) 93:372-6.

130. Wu et al., Markers of inflammation and risk of ovarian cancer in Los Angeles County, 2009. Int J Cancer (2009) 124:1409-1415.

131. Wu et al., African Americans and Hispanics remain at lower risk of ovarian cancer than non-Hispanic Whites after considering non-genetic risk factors and oophorectomy rates. Cancer Epidemiol Biomarkers Prev (2015) 24:1094-1100.

132. Zhou et al., Pelvic inflammatory disease and the risk of ovarian cancer: a meta-analysis. Cancer Causes Control. (2017) 28(5):415-428.

APPENDIX A

CURRICULUM VITAE
University of Alabama at Birmingham
School of Medicine Faculty

Date: January 30, 2019

PERSONAL INFORMATION

Name: Michael J. Birrer, MD, PhD
Citizenship: US
Foreign Language(s): None
Home Address: 2024 2nd Avenue N., Unit #1601, Birmingham, AL 35203
Telephone: 617-320-7460 (cell)

RANK/TITLE

Department: Director, Comprehensive Cancer Center
Professor of Medicine, Division of Hematology-Oncology
Professor of Pathology, Obstetrics and Gynecology
Business Address: UAB Comprehensive Cancer Center
1824 Sixth Avenue South, WTI 202
Birmingham, AL 35294-3300
Phone: 205-996-2524
Fax: 205-975-7428
Email: mbirrer@uab.edu

HOSPITAL AND OTHER (NON ACADEMIC) APPOINTMENTS
PROFESSIONAL CONSULTANTSHIPS

8/1/2017-Present	Director, Comprehensive Cancer Center
11/1/2008-7/31/2017	Director, Medical Gynecologic Oncology Director, Gynecologic Cancer Research Program Gillette Center for Gynecologic Oncology, Massachusetts General Hospital Leader, Dana-Farber/Harvard Cancer Center Gynecologic Cancer Program
7/1/2000-10/31/2008	Deputy Branch Chief, Cell and Cancer Biology Branch, CCR, NCI
7/1/1991-10/31/2008	Chief, Molecular Mechanisms Section, Center for Cancer Research, National Cancer Institute
1988-1995	Medical Oncology Consultant to Gynecologic Oncology Tumor Board, National Naval Medical Center
1988-2008	Attending Physician, National Naval Medical Center
1988-2008	Attending Physician, Clinical Center, National Cancer Institute (NCI), Bethesda

EDUCATION

Year	Degree	Institution
8/73-6/76	BS	Rensselaer Polytechnic Institute, Troy, NY Major - Biology, Minor - Philosophy, GPA 3.99 cum Laude
7/76-6/82	MD	Albert Einstein College of Medicine, Bronx, NY
7/76-6/82	MS, PhD	Albert Einstein College of Medicine, Bronx, NY Microbiology and Immunology

Thesis: The Role of Measles Virus in Multiple Sclerosis
Mentor: Dr. Barry Bloom, Chairman, Department of Microbiology and Immunology

MILITARY

U.S. Public Health Service (Serial No. 59928)

1991	Lieutenant Commander 04
1992	Commander 05
1997	Captain 06 Temporary Grade
2001	Captain 06 Permanent Grade

LICENSURE

1985	Medical License: Maryland
1982	Medical License: Massachusetts #52635

BOARD CERTIFICATION

1982	National Board of Medical Examiners
1985	Diplomate, American Board of Internal Medicine
1987	Diplomate, Subspecialty of Medical Oncology

POSTDOCTORAL TRAINING

7/1/1982-6/30/1983	Internship, Internal Medicine, Massachusetts General Hospital, Boston, MA (Dr. John Potts, Chairman, Department of Medicine)
7/1/1983-6/30/1985	Residency, Internal Medicine, Massachusetts General Hospital, Boston, MA
7/1/1985-6/30/1988	Fellowship, Medical Oncology, Medicine Branch, Clinical Oncology Program, National Cancer Institute, Bethesda, MD (Dr. Robert C. Young, Associate Director, Clinical Oncology Program)

ACADEMIC APPOINTMENTS

Year	**Rank/Title Institution**
8/1/2018 – Present	UAB appointment to Level II Graduate Faculty
8/1/2017 – Present	Evalina B. Spencer Chair in Oncology
8/1/2017-Present	Professor of Medicine, University of Alabama at Birmingham Division of Hematology-Oncology
11/1/2008-7/31/2017	Professor, Department of Medicine Harvard Medical School, Boston, MA
7/1/1991-10/31/2008	Senior Investigator, National Cancer Institute, NIH
7/1/1988-6/30/1991	Investigator, National Cancer Institute, NIH
1988-1995	Assistant Professor, Uniformed Services University of Health Sciences, Department of Medicine, Naval Hospital, Bethesda, MD
1982-1985	Instructor in Medicine, Harvard Medical School, Boston, MA

AWARDS/HONORS

1973-1976	Dean's List (all semesters)
1976	Phi Lambda Epsilon
1976	Sigma Xi Award

1977-1982	Medical Scientist Training Program (5T32GM7288)
1980	Alpha Omega Alpha, National Medical Honorary Society
1988	Outstanding Performance, Uniformed Services University of Health Sciences
1992	Division of Cancer Prevention and Control Employee of the Month
1992	Public Health Service Achievement Award
1993	Public Health Service Citation
1994	Equal Employment Opportunity Officer's Achievement Award
2010-2016	Best Doctors in America
2010-2016	Top Doctors in Boston, Boston Magazine
2014	Public Service Award, Foundation for Women's Cancer
2015	Director's Service Award, NCI
2016	Claudia Cohen Research Foundation Prize for Outstanding Gynecologic Cancer Researcher

PROFESSIONAL SOCIETIES/ MEMBERSHIPS

1986-1988	American Association of Clinical Oncology
2003-present	American Association of Clinical Oncology
1986-present	American Association for Cancer Research
1989-present	Gynecologic Oncology Group
2001-present	Society of Gynecologic Oncologists
2007-present	International Gynecologic Cancer Society
2011-present	European Society of Medical Oncology

COUNCILS AND COMMITTEES

1988-1991	Clinical Oncology Program, Fellowship Selection Committee, NCT, NCI
1990-1993	Committee for the Protection of Human Subjects, NNMC, Bethesda, MD
1990-1993	Grant Review for Early Detection and Community Oncology Program, DCPC, NCI
1990-2013	Experimental Medicine Committee, Gynecologic Oncology Group (GOG)
1992-present	Division of Cancer Prevention and Control Fellowship Selection Committee, DCPC, NCI
1992-1994	NCI Institutional Review Board for Extramural Affairs, NCI, Bethesda, MD
1993	Planning Committee for International Conference for Colorectal Screening
1994	Chairman, International Conference for Colorectal Screening: New Technology Development Section
1994	Source Evaluation Group for Early Detection Network, DCPC, NCI
1995	Committee for Scientific Diversity, NIH
1995-1999	Medical Oncology Consultant to Gynecologic Oncology Tumor Board, WRAM
1997-2001	Ovarian Cancer Research Program, Department of Defense (DOD)
2000-2001	Chair – Ovarian Cancer Research Program, DOD
2001	Steering Committee, Gynecologic Oncology Faculty, CCR
2001	Pre-Clinical Working Group
2001-2002	Chair Emeritus, Ovarian Cancer Research Program, Intergration Panel DOD
1998-2013	Protocol Development Committee, GOG
2001-2013	Ovarian Committee, GOG
2001-2013	Ancillary Data Committee GOG
2001-2002	Co-Chair, Committee for Experimental Medicine GOG

2002	Gynecologic Cancers Progress Review Group Pre-Meeting with Director
2002	Gynecologic Cancer Progress Review Group Implementation Meeting
2002-present	Chair, Committee for Experimental Medicine GOG
2002-2004	American Society of Clinical Oncology (ASCO) Program Committee Gynecologic Cancer Track
2002-2005	SGO Program Committee Member
2003	GOG Site Visit Committee for Experimental Medicine (priority score 175)
2003	Chair GOG Corporate Scientific Symposium
2003-present	Chairman's Advisory Committee GOG
2004	Member GOG Corporate Scientific Symposium Committee
2005	Program Committee, International Meeting on Ovarian Cancer
2005-2016	Co-Chair, Gynecologic Cancer Steering Committee (GCSC) of the National Institute of Health (NCI)
2006-present	Member, Reproductive Scientific Development Program Board
2009-2011	Chair Translational Science Committee, Gynecologic Cancer Intergroup (GCIG)
2009-2013	TCGA Steering Committee
2009-2016	Co-Chair of the Gynecologic Cancer Steering Committee (GCSC) of the National Institute of Health (NCI)
2009-present	Scientific Board, Target Ovarian Cancer
2010-present	Program for the Assessment of Clinical Cancer Tests (PAACT)
2010-2012	Medical Advisory Board, Illumia
2010-present	Witherspoon Council on Ethics and the Integrity of Science
2011-2014	Member, National Clinical Trial Network Working Group
2011-present	External Advisory Board Clinical Proteomic Tumor Analysis Consortium (CPTAC)
2012-2015	ASCO Program Committee Tumor Biology Track
2012-present	Group Banking Committee
2013-present	Co-Chair, Tanslational Research Working Group, NRG
2013-present	NRG Research Committee
2014-2015	Clinical Trial Planning Meeting Organizing Committee – Uterine Corpus
2014-2015	Co-chair UPSC CTPM Group 2
2014-present	Chair, NCTN Correlative Science Committee
2015-present	Immunogen GYN Steering Committee for Development of IMGN853
2015-present	Member, Special Commission on Ovarian Cancer, Massachusetts Department of Public Health
2017-2018	SGO Program Committee Member
2018	Cancer Research UK Science Committee Expert Review Panel
2018-present	AL Governor appointment to the Study Commission for Gynecologic Cancers
2018	NCI ZRG1 OTC-W Study Section Review Panel
2018	Review of NCI Intramural Research Programs

UNIVERSITY ACTIVITIES

2000-present	External Advisory Board, MD Anderson Cancer Center Uterine SPORE
2006	Member, Marsha Rivkin Center for Ovarian Cancer Research Grant Review
2008-2013	Chair, External Advisory Board Fox Chase Cancer Center Ovarian SPORE
2008-2017	Member, Cancer Center Leadership Committee, MGH
2009-2013	External Advisory Board, Stanford University Ovarian SPORE

2010-present	Scientific Board, Terry Fox Research Institute - Biomarker Program 'COEUR'
2011-present	External Advisory Board Roswell Park Ovarian SPORE
2012-2017	Member, Academic Advisory Group, MGH
2014-present	Member, Marsha Rivkin Scientific Board

EDITORIAL BOARDS

Disease Markers
American Journal of Obstetrics and Gynecology
Women's Health Journal
Clinical Cancer Research
Journal of Biologic Chemistry
Journal of the National Cancer Institute – Associate Editor

EDITORIAL REVIEW

American Journal of Respiratory Diseases, Biochim Biophysis Acta, Blood, Cancer, Cancer Research, Gynecologic Oncology, Journal of Clinical Oncology, Journal of the National Cancer Institute, Journal of Obstetrics and Gynecology, Molecular Carcinogenesis, Molecular and Cellular Biology, Oncogene, Proceedings of the National Academy of Sciences, National Science Foundation Grant Review, The Israel Science Foundation

MAJOR RESEARCH INTERESTS

My major research interest is in characterizing the genomics of gynecologic cancers and translating it into improving in the clinical management of these diseases. The research focuses on the development of early detection assays, elucidation of new biology, discovery of novel therapuetic targets, and identification and validation of predictive and prognostic biomarkers. It is our vision that through a better understanding of the molecular underpinnings of these cancers, we will be able to better diagnose and treat these diseases.

TEACHING EXPERIENCE

1988-1995	Medical Student Preceptor, Uniformed Services University of Health Sciences
1991-1993	Lecturer, Clinical Service Lecture Series, NMOB, DCT, NCI
1991-present	Lecturer, Cancer Prevention Fellowship Seminar Series, DCPN, NCI
2010-present	Gynecologic Cancer Academy
2015-present	Member, Thesis Committee MIT Graduate Student

RESEARCH TRAINEES

Name	**Position**	**Position Obtained after Training**
Richard Rosenberg, MD	Clinical Associate	Assistant Professor, University of Arizona
Dennis Sanders, MD	Clinical Associate	Assistant Professor, Boston University
Eva Szabo, MD	Clinical Associate	Senior Investigator, NCI
Powel Brown MD, PhD	Clinical Associate	Associate Professor, Dept. of UTS
Steven Lemon, MD	Clinical Associate	Assistant Professor, Creighton University
Anita Sabichi, MD	Clinical Associate	Assistant Professor, MDACC
Rhoda Alani	Howard Hughes Scholar	Massachusetts General Hospital
Michael Teneriello, MD	Gyn Oncology Fellow	Assistant Professor, University of Pittsburgh
Robert Taylor, MD	Gyn Oncology Fellow	Assistant Professor, USUHS
Sung Kim, MD	Fogarty Fellow	Assistant Professor, Seoul Korea

Hiro Dosaka	Fogarty Fellow	Assistant Professor, Sapporo, Japan
Mary Parker, MD	Gyn Oncologist	Tripler Army Base Ha.
Kelly Gendreau	Student Trainee	Colgate University
Tricia Francis	Student Trainee	University of Michigan
Julie Francis	Student Trainee	Harvard University
Daniel Gephart	Student Trainee	University of Michigan
Yatia Gross	Student Trainee	McKinley High School
Achim Moesta	Student Trainee	Colgate University
Robert Kao	MCPS/HHMI	Boston College
Ginger Gardner, MD	GCF Scholar	Mt Sinai
Denver Hendricks, PhD	Fogarty Fellow	Research Associate UCT S.A.
Kristin Zorn, MD	GCF Fellow	University of Oklahoma
Jeff Chick	Colgate Univerity	Colgate University
Will Winter, MD	Gyn Oncology Fellow	Brooks Army Hospital

MAJOR LECTURES AND VISITING PROFESSORSHIPS

1988 Grand Rounds, Clinical Oncology Program, NCI, Bethesda, MD

Grand Rounds, Fox Chase Cancer Center, Philadelphia, PA

1991 *Malignancies of the Aerodigestive Tract,* ICN-UCLA Symposium: Keystone, CO

Department of Head & Neck Cancers, M.D. Anderson Cancer Center, Houston, TX

1992 *Early Detection of Cancer: Challenges for Molecular Biology*, Early Detection Branch, DCPC, NCI, Gaithersburg, MD

Microbiology and Immunology Sem. Series, Medical College of Virginia, Richmond, VA

1993 Laboratory and Branch Chiefs Meeting, DCPC, NCI

Grand Rounds, Clinical Oncology Program, NCI, Bethesda, MD

1994 DCPC Colloquim

1995 *Novel Intervention Agents*, Chairperson, Mini-symposium, AACR, Washington, DC

1996 Department of Biochemistry, University of Cincinnati, Cincinnati, OH

Cancer Symposium, CHEP, Perry Point, MD

1997 Grand Rounds, University of Illinois, Chicago, IL

13th Annual Ella T. Grasso Memorial Conference, University of Connecticut, Farmington, CT

2001 Gynecologic Cancer Translational Research Retreat, April

2001 *Molecular Prevention Course Oncogenes and Tumor Suppressor Genes,* August

2001 Co-Chair Gynecologic Oncology Faculty Retreat

2002 *Director's Challenge,* PI Meeting Bethesda MD, Nov 6-8

2002 *RTK Inhibitors,* Chair, ASCO Molecular Therapeutics Symposium, San Diego, CA, Nov 8, 2002

International Meeting for Early Ovarian Cancer, Dulles Airport, Washington, D.C.

2003 Chair, Thesis Defense Committee Department of Gynecologic Oncology, University of South Florida, Tampa, FL

Ovarian Cancer Biology, Survivor's Course, Society of Gynecologic Oncology Annual Meeting, Miami, FL

Expression Profiling of Ovarian Cancer, Oncogene Meeting, Frederick, MD

Novel Retinoids, Hormonal Carcinogenesis Faculty Meeting, Rocky Point, MD

Expression Profiling of Ovarian Cancer, 19th Annual Ella T. Grasso Memorial Conference, University of Connecticut, Hartford, CT

2004 *Expression Profiling of Ovarian Cancer,* Ovarian Cancer Mouse Models Meeting, MMHCC San Juan, Puerto Rico

Expression Profiling of Ovarian Cancer, 1st International, Conference on Ovarian Cancer, Physician Education Resource Co-Organizer, Park Plaza Hotel, New York, NY

Molecular Classification of Ovarian Cancer, Johns Hopkins School of Medicine, Department of Pathology, Baltimore, MD

GOG-SPORE Collaborations, SPORE Annual Meeting, Baltimore MD

Genomic Analysis of Ovarian Cancer, Division of Gynecologic Oncology, University of Alabama, Birmingham, AL

Gynecologic Cancer Intergroup Presentation to the NCI Director, Bethesda, MD

Genomic Comparison of Animal and Human Ovarian Cancer, International Society of Gynecologic Cancer, Edinburgh, Scotland, UK

Preparing for Peer Review Ovarian Cancer Ovarian Cancer National Alliance Washington, DC

NCI Initiatives, Gynecologic Cancer Foundation Allied Health Group Presentation SGO Annual Meeting, Miami, FL

Ovarian Cancer Biology, Survivor's Course, Society for Gynecologic Oncologists Annual meeting, Miami FL

2005 Moderator and Co-organizer, Translational Science Session, 2nd International Conference on Ovarian Cancer, Plaza Hotel, New York, New York

ET-743- a new cytotoxic agent, 2and International Conference on Ovarian Cancer, Plaza Hotel, New York, NY

Molecular Profiling of Ovarian Cancer Genomics Mini-symposium AACR, Anaheim, CA

GOG Priorities "Omics" Corporate Symposium Gynecologic Oncology Group Semi- Annual Meeting, Baltimore, MD

SPORE Investigator Meeting, Co-Chair, Ovarian Break-Out Group

Co-organizer State of the Science Meeting, Ovarian Cancer, Rockville MD

Moderator, Biomarkers and Phase III Trial Design, State of the Science Mtg., Ovarian Cancer, Rockville, MD

Molecular Etiology, Ovarian Cancer Session, Symposium on Women's Cancer, King Hussein Cancer Center, Amman, Jordan

Chair, Ovarian Cancer Treatment Session, King Hussein Cancer Center, Amman, Jordan

Tissue Based Assays-Con Ovarian Cancer National Alliance Annual Meeting, Atlanta, GA

State of the Science Summary, Ovarian Cancer National Alliance Annual Meeting, Atlanta, GA

The Genomic Analysis of Ovarian Cancer, Ovarian Cancer Symposium, Massachusetts General Hospital Cancer Center, Boston, MA

The Genomic Analysis of Ovarian Cancer, Obstetrics and Gynecology Grand Rounds, Magee Women's Hospital, Pittsburgh, PA

Characterization of a gene signature, which predicts survival in patients with advanced stage, high grade ovarian cancer. Research Seminar, Magee Women's Hospital, Pittsburgh, PA

The Genomics Analysis of Ovarian Cancer: what does it tell us? University of Texas at San Antonio, San Antonio, TX

State of the Science Summary, Gynecologic Cancer Intercrop, Paris, France

2006 GOG Scientific Symposium, *Angiogenesis: A new therapeutic target.* Symposium Chair, San Diego, CA

SPORE-GOG collaborations, Co-Chair SPORE mid-winter meeting, Houston, TX

Whole-Genome Expression Profiling of Papillary Serous Ovarian Cancer: Activated Pathways, Potential Targets and Noise Tri Medicine Meeting, Cambridge Health Technology, San Francisco, CA

Tumor infiltrating lymphocytes in ovarian cancer, Data Club, Cell and Cancer Biology Branch, Bethesda, MD

Focused Plenary Session*: Basic Science and Translational Medicine,* Chair, Society of Gynecologic Oncology Annual Mtg Palm Springs, CA

Translational Science in GOG, Sunrise Session 6: Gynecologic Oncology Group (GOG) Update: What's New in 2006, Society of Gynecologic Oncology Annual Mtg Palm Springs, CA

The Odyssey of Target Identification, Express Postgraduate Course 3: "Targeting Targeted Therapy in Gynecologic Cancers", Society of Gynecologic Oncology Annual Mtg, Palm Springs, CA

Identification of cJun/AP-1 target genes by microarrays and chromatin immunoprecipitation assays: clues to its diverse biologic activities TOIG Seminar, Medical Board Room, Bethesda, MD

Genomic Analysis & The Pathogenesis of Ovarian Cancer Lynn Cohen Foundation Meeting, New York School of Medicine, New York, NY

Translational Science Session, Moderator, Third International Conference on Ovarian Cancer Roosevelt Hotel, New York, NY

Genomic Analysis and Ovarian Cancer Translational Science Session, Third International Conference on Ovarian Cancer Roosevelt Hotel, New York, NY

Disease Classification, 1st International Conference on Ovarian Cancer, Aegean Conference, Crete

` *Identification of important signaling pathways in serous tumors of the Ovary* kCon/fab Annual Meeting, Couran Cove, Australia

Oncogenes and Tumor Suppressor Genes Honors Lecture, University of Cape Town, Cape Town, South Africa

Identification of important signaling pathways in serous tumors of the ovary: a genomic analysis. University of Cape Town, Cape Town, South Africa

Intramural Research Program, Ovarian Cancer National Alliance Annual Meeting, Washington DC

Diagnostics & Imaging in the Management of Ovarian Cancer Second Annual World Oncology Congress, Marriott Marquis, New York, NY

Profile Outcomes Prediction, Second Annual World Oncology Congress, Marriott Marquis, New York, NY

Molecular Biology Session, Chair, State of the Science Mtg Endometrial Cancer, Manchester, UK

Drug Discoveries, 7th Annual International Conference on Ovarian Cancer, MDACC, Houston TX

2007 *LMP/Low grade tumors: are they a unique entity? Molecular Biology*Scientific Symposium, GOG Semi-annual Meeting San Diego, CA

Expression Profiling of Ovarian Cancer: New Insights into its Origin and Novel Therapeutic Targets. Susan Klein Kamen Endowed Lectureship, Memorial Sloan-Kettering Cancer Center, Department of Medicine, NewYork, NY

New updates from the Ovarian SPOREs, Chair, Post Graduate Session, Society of Gynecologic Oncologists Annual Meeting, San Diego, CA

Expression Profiling of Ovarian Cancer: New Insights on the Origin and Treatment of the Disease, Beijing Symposium: Cell Signaling in Cancer, Development and Stem Cells, Beijing, China

Expression Profiling of Ovarian Cancer: New Insights on the Origin and Treatment of the Disease, The Chinese University of Hong Kong Department of Obstetrics and Gynecology, Hong Kong

SPORE Gynecologic Cancer Breakout Session, Moderator

Expression Profiling of Ovarian Cancer: New Insights on the Origin and treatment of the Disease, Seminar, Massachusetts General Hospital, Boston, MA

Translational Research in Ovarian Cancer: Gynecologic Oncology 5th Group, 5th Korean Gynecologic Oncology Group Meeting, Seoul Korea

Genomic Profiling of Ovarian Cancer: New Insights into Pathobiology, Korean Society of Gynecologic Cancer, Seoul, Korea

2008 *Tumor Assessment*, Targeted Therapies, GOG Symposium, Moderator

Genomics and Proteomics: The Future is Now, GOG Scientific Symposium, Moderator, GOG

Semi-Annual Meeting, San Diego, CA

Genomic Analysis of Ovarian Cancer, Cancer Center Grand Rounds, Massachusetts General Hospital, Boston MA

Genome-Wide Target Discovery in Ovarian Cancer, Keynote Presentation, 1st Ovarian Cancer Action International Conference, London, UK

Moderator, 1st Ovarian Cancer Action International Conference, London, UK

Moderator, Post Graduate Course 2: What's new in the Gynecologic SPOREs, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

Genomic Approaches to Target Discovery, Post Graduate Course 4, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

The Genomic Revolution: How it will Change the State of the Science of Ovarian Cancer! State of the Science Meeting, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

Other Novel Strategies Targeting Cellular Pathways in the Treatment of Advanced Ovarian Cancer, SGO Satellite Symposium, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

The Biology of Ovarian Cancer, Survivor's Course Gynecologic Cancer Foundation, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

Cancer Stem Cell Gene Signature identified from ovarian tumor side populations, Focused Plenary on Ovarian Cancer, Society of Gynecologic Oncologists, Annual Meeting, Tampa, FL

Genomic Analysis of Ovarian Cancer: where will it lead us? Bench to Bedside AACR Special Meeting, Amman, Jordan

New Agents and Translational Research in Ovarian Cancer Moderator, Fifth International Symposium on Ovarian Cancer and Gynecologic Malignancies New York, NY

Optimal Systemic Therapy for Advanced Uterine Cancer Moderator, Fifth International Symposium on Ovarian Cancer and Gynecologic Malignancies New York, NY

Therapy for a Patient with Platinum Resistant/refractory Recurrent Ovarian Cancer should be selected Based on Result of an In Vitro Extreme drug Sensitivity/Resistance Assay- Con, Fifth International Symposium on Ovarian Cancer and Gynecologic Malignancies New York, NY

Biobehavioral influences on tumor biology: Preclinical models of neuroendocrine regulation, Symposium organizer, Natcher Auditorium, NIH, Bethesda, MD, June 24, 2008

Clinical and Translational Research Opportunities to Expand the Paradigms Biobehavioral

influences on tumor biology: Preclinical models of neuroendocrine regulation, Natcher Auditorium, NIH, Bethesda, MD, June 24, 2008

The Molecular Classification of Ovarian Cancer: survival, resistance, and new targets. 2nd International Conference on Ovarian cancer: State of the Art and Future Directions, Rhodes, Greece June 26, 2008

Ovarian Cancer – Update of Research, Ovarian Cancer National Alliance, Omni Shoreham Hotel, Washington D.C., July 9, 2008

Chaired Session: Translational Science in Gynecologic Cancer, The 12th International Gynecologic Cancer Society, Dusit Thani Hotel, Bangkok, Thailand, October 25 – 28, 2008

Frontiers: Translational Science in Gynecologic Cancer, The 12th International Gynecologic Cancer Society, Dusit Thani Hotel, Bangkok, Thailand, October 25 – 28, 2008

The Biology of Early Ovarian Cancer, GYN-Onc Research Program, Intramural Research Program, NCI, NIH, Bethesda, MD

Survivors Course GCF, Hughes Auditorium, Chicago, Illinois, July 2008

Research Updates, Part 1, Ovarian Cancer National Alliance, Omni Shoreham, Washington, DC, July 8, 2008

2009 EOCPP External Advisory Committee Meeting, MSKCC, New York, NY, January 29, 2009

Biological Markers; New Markers for Response, 7th International Symposium Advanced Ovarian Cancer: Optimal Therapy, Update, Valencia, Spain, February 27 & 28, 2009

The Genomics of Ovarian Cancer: What does it tell us? Moreton Grand Rounds, University of Mississippi, Jackson, MS, March 10, 2009

Personalized Medicine: Clarity or Confusion? Alpha Omega Alpha Banquet, University of Mississippi, Jackson, MS, March 10, 2009

Ovarian Congress, New York, NY, March 20-21, 2009

The Genomics of Ovarian Cancer: What does it tell us? Cancer Center Grand Rounds, The Cancer Institute of New Jersey, New Brunswick, NJ, April 8, 2009

New Insights in the Biology and Pathogenesis of Epithelial Ovarian Cancer Gene Expression Profiles in Ovarian Carcinoma in Relation to Phenotype, Chemosensitivity and Prognosis, Nordic Society of Gynecologic Oncology, Stockholm, Sweden, April 23 & 24, 2009

NCRN/GCIG NCI Clinical Trials Planning Meeting, Manchester, England, June 17-19, 2009

GOG/Committee of Experimental Medicine, Chair GOG/Scientific Session Moderator, Baltimore, MD, July 16-19, 2009

The Biology for Ovarian Cancer GOG/Survivors Course, Baltimore, MD, July 16-19, 2009

Stromal–Epithelial Interactions in Ovarian Cancer: Implications for Biology and Treatment, Australia Ovarian Cancer Study, Familia Aspects of Cancer, Cancer and Families: Research and Practice, Queensland, Australia, August 11-14, 2009

Ovarian Cancer, Annual Community Oncology Research Forum, Dallas, Texas, September 11, 2009

Session Debate (Con): All Patients at High Risk for Ovarian Cancer Should Undergo Routine Screening; Session Debate (Con): Therapy for the Patient with Platinum-Resistant/Refractory Recurrent Ovarian Carcinoma Should be Selected Based on the Results of an In Vitro Extreme Drug Sensitivity/Resistance Assay, Fifth Annual Oncology Congress, San Francisco, CA, September 25, 2009

Novel Therapeutics in Clinical Trial, American College of Radiology Imaging Network, Arlington, VA, October 2, 2009

The Biology of Ovarian Cancer, Ovarian Cancer Survivors Course, Gynecologic Cancer Foundation, Washington, D.C., November 7, 2009

GOG Site Visit, Bethesda, MD, November 9-10, 2009

First Global Workshop on Ovarian Cancer, Chicago, IL, November 20 & 21, 2009

2010 *USON GYN CME Meeting,* Phoenix, AZ, January 15-16, 2010

Canadian Ovarian Cancer Research, Toronto, Canada, February 4, 2010

Future Directions in Ovarian Cancer, London, UK, February 11, 2010

The Program for the Assessment of Clinical Cancer Tests (PACCT/CADP) Chicago, IL, April 7, 2010

Stand Up to Cancer/Phosphatidylinositol 3'-Kinase Dream Team, Walter E. Washington Convention Center, Washington, DC, April 16-18, 2010

Gynecologic Malignancy Advisory Board, New York, NY, May 21 & 22, 2010

What are promising targets for future therapeutic approaches? 4th Ovarian Cancer Consensus Conference, Vancouver, Canada, June 23-27, 2010

The Biology of Ovarian Cancer, Ovarian Cancer Survivors Course, Boston, MA, July 15, 2010

The GOG Specimen Bank and Translational Research, GOG Summer Scientific Session, Boston, MA, July 15-18, 2010

DNA Repair, Cancer Education Consortium Annual Workshop (CEC), Lansdowne Conference Center, Leesburg, VA, September 13, 2010

PARP Inhibitors and BRCA 1 / 2 Associated Ovarian Cancers, Ella Grasso Memorial CME Conference, Yale University, New Haven, CT, November 17, 2010

Emerging Therapeutics in Recurrent Disease, SGO Session III, Northwestern University, Chicago, IL, December 4, 2010

2011 *P53 in ovarian cancer and how it might relate to breast cancer.* Breast Cancer Grand Rounds, Massachusetts General Hospital, Boston, MA, January 4, 2011

Innovative Treatments in Ovarian Cancer, Annual Meeting of the Israeli Society for Clinical Oncology and Radiation Therapy, Tel Aviv, Israel, January 12-14, 2011

Innovative Treatment in Ovarian Cancer - Current Status and Future Prospective, Israeli Gynecologic Oncology Society, Tel Aviv, Israel January 12-14, 2011

The Post Cancer Genomic Atlans Era - Where Do We Go from Here? Helene Harris Memorial Trust 12th International Forum on Ovarian Cancer, Miami, FL January 15-19, 2011

GYN Cancer Academy Educational Program, Inaugural Mentor's Meeting, Valencia, Spain, March 31, 2011

Canadian Ovarian Cancer Resources, Montreal, Canada, April 8, 2011

Ovarian Cancer: The Origin and New Therapeutic Targets, Wayne State University Grand Rounds, Detroit, MI, May 24, 2011

A Phase II Trial of Iniparib (BSI-201) in combination with gemcitabline/carboplatin (GC) in patients with platinum-sensitive recurrent ovarian cancer, ASCO Annual Meeting, Chicago, IL, June 3-7, 2011

When is a Predictor Clinically Useful? Omics Meeting, Bethesda, MD, June 23 & 24, 2011

Ovarian Cancer Genetics, Ovarian Cancer Survivors Course, Boston, MA, August 20, 2011

Biomarkers for Ovarian Cancer: Where can they help? Early Detection Research Network (EDRN), Bethesda, MD, September 13-15, 2011

DNA Repair, Cancer Education Consortium, Leesburg, VA, September 25 & 26, 2011

Appointment Panel, Cancer Research, London, UK, September 29, 2011

Ovarian Clinical Trials Planning Committee, Philadelphia, PA, November 2, 2011

Future/Emerging Treatments? Ovarian Cancer Survivor's Course, Houston, TX, December 1, 2011

2012 *New Strategies to Identify and Screen Women at Risk for Ovarian Cancer,* GOG Scientific Session, San Diego, CA, January 26-28, 2012

Ovarian Cancer: State of the Science Past, Present and Future, Baylor-MD Anderson Joint Symposium, MD Anderson, Houston, TX, February 3, 2012

Improving Outcomes for Woman with Advanced Ovarian Cancer, GOG-0218, Prague, The Czech Republic, February 11-12, 2012

The Future of Ovarian Cancer Management: Biomarkers in Oncology, Avastin Ovarian Cancer Launch, Prague, The Czech Republic, February 11-12, 2012

The Challenges of Inter and Intra-Tumoral Hetergenicity in the Management of Ovarian Cancer, American Association for Cancer Research (AACR), Chicago, IL, April 2, 2012

Ovarian Cancer: State of the Science, Beth Israel Deaconess Medical Center Grand Rounds, Boston, MA, June 13, 2012

Targeting Genomic Chaos in Gynecologic Oncology, Program Chair for GOG Summer 2012 Scientific Session, 85th Semi-Annual Meeting, Boston, MA July 26, 2012

Recent Advances in Ovarian Cancer Clinical Research, Keynote Speech*; Everything You Ever Wanted to Know about PARP Inhibitors but were Afraid to Ask,* 9th Biennial Ovarian Cancer Research Symposium, Marsha Rivkin Center for Cancer Research, Seattle,WA, September 7, 2012

2013 National Cancer Institute (NCI), National Institutes of Health (NIH), U.S. Department of Human Services, NCI Precision Cancer Medicine Working Group, Bethesda, Maryland, January 10, 2013

Report and discussion of assays to guide treatment, Defining Clinical Utility of Molecular Diagnostics for Cancer Treatment, PACCT Meeting, Bethesda, MD, January 11, 2013

Ovarian Cancer Management-On the Front Line, Chair, Scientific Session, GOG Semi-Annual Meeting - San Diego, CA, January 24-27, 2013

Genomic analysis. Keynote Lecture, 9th International Symposium on Advanced Ovarian Cancer: Optimal Therapy, Valencia, Spain, March 2, 2013

Trials and Tribulations of Ovarian Cancer Screening; Early Detection of Ovarian Cancer: Old

Trials and New Biology; 8th EDRN Meeting, Scientific Program Committee Meeting, Bethesda, MD, March 13-14, 2013

Shift in the Treatment Paradigm of Ovarian Cancer, Chennai, Hyderabad, Hyderabad/Mumbai and Mumbai, India, Global Oncology Summit, March 29-April 1, 2013

NCI Group Banking Committee (GBC) Face-to-Face Meeting, Columbus, OH, April 18-19, 2013

The Importance of Correlative Studies in Clinical Trials Translational/Anti-Angiogenesis, 13th Annual Continuing Professional Development Meeting, Toronto, Ontario, April 26, 2013

The Center for the Study of Technology and Society, Witherspoon Council Meeting, 1) contemporary genetics and 2) end-of-life issues, Washington, D.C., May 28, 2013

Chromatin Regulatory Mechanisms in Ovarian Cancer, External Advisory Committee Meeting, Wistar Institute, Philadelphia, PA, May 29, 2013

The Future of Ovarian Cancer Treatment: Personalized Medicine, Ovarian Cancer Survivors Course, New York, NY, June 8, 2013

Gynecologic Cancer Trial Portfolio Presentation, (Ovarian and Corpus/Cervix Presentations) NCI, NCTN Summer Working Group Meeting, Rockville, MD, July 1-2, 2013

State of the Science in Cervical Cancer: Where We Are Today and Where We Need to Go, GOG 87th Semi-Annual Meeting, San Antonio, TX, July 18-20, 2013

GYN Translational Research Organization in Europe, 2nd Gynaecological Cancer Academy (GCA) Workshop, Paris, France, September 4-9, 2013

Harnessing Genomic Data to Guide Proteomic Analysis: Can Expression Profiling Identify Early Detection Biomarkers? 26th EDRN (Early Detection Research Network) Steering Committe Meeting/Data Jamboree, Seattle, WA, September 10-12, 2013

Target Ovarian Cancer Scientific Advisory Board, Discussion Leader, London, UK, October 17, 2013

Current and Emerging Biomarkers and Targets in Ovarian Cancer, 18th International Society of Gynaecological Oncology (ESGO), Liverpool, UK, October 18-19, 2013

Future of Ovarian Cancer Treatment: Personalized Medicine, Foundation for Women's Cancer, Ovarian Cancer Survivors Course, Washington, DC, November 1-2, 2013

Translational Research Report, GOG Chairman's Working Group, Philadelphia, PA, November 4, 2013

Ovarian Cancer: The State of the Science, Grand Rounds, Karmanos Cancer Institute, Detroit MI,

November 21, 2013

Genetic Profiling Uncovers New Therapeutic Approaches to Ovarian Cancer, 22nd Annual Meeting MITO, "From the Macroscopic to the Microscopic: The Evolution in All Treatments for Ovarian Cancer", Rome, Italy, November 28-29, 2013

2014 GOG/NRG Oncology Semi-Annual Meeting, San Diego, CA, February 6-9, 2014

Clinical Experience with the Combination of Pimasertib and SAR245409, Opening Remarks, Merck Serono & Quintiles Investigator Meeting, MEKi 012 Study Protocol, Rome, Italy, March 5-7, 2014

SGO 45th Annual Meeting on Women's Cancer, March 22 & 23, 2014 Foundation for Women's Cancer, Board and Business Meeting, Board of Directors Meeting and receipt of the 2014 Public Service Award, Tampa, FL, March 23, 2014

National Institutes of Health, National Cancer Institute Think Tank, Bethesda, MD, April 16, 2014

Personalized Medicine: Are All Cancers the Same? Foundation for Women's Cancer, Gynecologic Cancer Survivors Course, Garden City, NY, May 2, 2014

Druggable Targets in Ovarian Cancer, Third Gynaecological Cancer Academy, Stresa, Italy, May 16-17, 2014

Osmotic Micro-Pump as Delivery System for Intraperitoneal Chemotherapy in the Treatment of Advanced Ovarian Cancer, DF/HCC and the Koch Institute, MIT, The Bridge Project Symposium and Networking Event. Award Announcement for team: Michael Cima, K1 Michael Birrer and Marcela Del Carmen. Cambridge, MA, May 22, 2014

Potential Biomarkers for FGFR inhibitor Therapy, May 31, 2014, Discussant; *Meta-Analysis of Public Microarray Databases for Prognostic and Predictive Gene Signatures of Late-Stage Ovarian Cancer,* Poster Presentation; *Molecular Profiling of Rare Uterine Tumors: A Potential Guide to Treatment?* Oral Presentation, ASCO Annual Meeting, Chicago, IL, May 30-June 2, 2014

Personalized Medicine and Cancer, OhioHealth Cancer Conference, Columbus, OH, June 7, 2014

Clinical Applications of the Cancer Gene Atlas (TCGA) in Endometrial Cancer, Nordic Society of Gynaecological Oncology Annual Meeting, Selfoss, Iceland, June 13-14, 2014

GOG/NRG Semi-Annual Meeting, Chicago, IL, July 10-12, 2014

Panel Moderator, "Pre-Operative Factors", American Brachytherapy Society, Gyn Multi-Disciplinary Summer Symposium, Chicago, IL, July 13, 2014

Welcome and Course Overview; New Therapeutic Agents in Ovarian Cancer: Foundation for

Women's Cancer, Gynecologic Cancer Survivors Course, Wyndham Hotel, Boston, MA, July 26, 2014

The Genomic Analysis of Ovarian Cancer: Where are We? The Wistar Institute, Philadelphia, PA, September 15, 2014

*Needs and Challenges in Ovarian Cancer; Overview of Bridge Project Research. "*Bridging the Gap in Ovarian Cancer", The Koch Institute for Integrative Cancer Research at MIT, Cambridge, MA, September 16, 2014

What are the Issues for Surgical Trials? Trials in Surgery Workshop; *Why Signatures Miss the Goal in Ovarian Cancer.* Fourth Gynaecological Cancer Academy, Frankfurt, Germany, September 19-20, 2014

Genomic Analysis of Ovarian Cancer: Where are We? Guest Lecturer, Richard W. TeLinde Lectureship, Grand Rounds, Johns Hopkins Hospital, Baltimore, MD, October 16, 2014

Panel Member, OncLive Peer Exchange for "Updates in Ovarian and Cervical Cancers" filming, Dallas, TX, October 18, 2014

Faculty Member, 10th Annual International Symposium on Ovarian Cancer and Gynecologic Malignancies "Medical Crossfire" filming, Dallas, TX, October 18, 2014

New Therapeutic Targets in Ovarian and Uterine Cancers, Chemotherapy Foundation Symposium: Innovative Cancer Therapy of Tomorrow, The Greenspan Meeting XXXII, Marriott Marquis, New York, NY, November 6, 2014.

Molecular Targeted Therapy for Ovarian Cancer, 30th Annual Ella T. Grasso Memorial Conference, Yale West Campus, Orange, CT, December 3, 2014

2015 *The Impact of Molecular Testing in Therapeutic Decisions in Ovarian Cancer,* 2015 Progress and Controversies in Gynecologic Oncology Conference, Barcelona, Spain, January 17, 2015

Curative Strategies for Primary Disease; Therapies for Driver Mutations Using Genomics to Stratify Patients or to Personalize Care, Ovarian Cancer Action, HHMT (Helen Harris Memorial Trust) 13th International Forum on Ovarian Cancer, Toledo, Spain, January, January 19, 2015

Genomic Analysis of Ovarian Cancer: Where Are We? Invited Speaker, Globeathon to End Women's Cancer, Inova Fairfax Hospital, Falls Church, VA, January 23, 2015

Genomic Expression Signatures to Predict Debulking Status, NRG Oncology Semi-Annual Meeting, Manchester Grand Hyatt, San Diego, CA, February 5, 2015

Endometrial Cancer Session: *From Histologic to Genetic Classification.* Ovarian Cancer Session: *Concepts in the Biology of Ovarian Cancer: The impact of Genetics on Clinical Practice***,** Pakistan Society of Clinical Oncology (PSCO) Gynecologic Cancer Symposium, Keynote Speaker and

Panel Member, Advancing Cancer Care, 2015 Women Cancer Meeting, "Current and Future Concepts", Lahore, Pakistan, February 14, 2015

Future of Ovarian Cancer Treatment: Personalized Medicine, Foundation for Women's Cancer, Ovarian Cancer Survivors Course, The Kravis Center, Cohen Pavillion, West Palm Beach, FL, February 28, 2015

The Molecular Genetics of Epithelial Ovarian Cancer, 10th Internatinal Symposium on Advanced Ovarian Cancer, Optimal Therapy Update, Valencia, Spain, March 6, 2015

Design a New Immunotherapy Trial in Ovarian Cancer: Phase I Plus Translational Research, Fifth Gynaecological Cancer Academy Workshop, Hotel Primus, Valencia, Spain, March 7, 2015

Personalizing Treatment of Ovarian Cancer: Has the Time Finally Arrived? Medscape Oncology Roundtable Filming, New York, NY, March 17, 2015

Ovarian Cancer, 29th Early Detection Research Network (EDRN) Steering Committee Meeting, Atlanta, GA, March 31, 2015

Global Gyn Steering Committee meeting for IMGN853, New York, NY, April 10, 2015

Antiangiogenetic therapy in gynecologic malignancies ~ prediction of efficacy, XXIV Scientific Meeting of the AGO, Salzburg, Austria, April 16, 2015

The Genomic and Epigenomic Characteristics of Long-Term survivors of Ovarian Cancer, 8th International Charite-Mayo-Conference, Berlin, Germany, April 18, 2015

The Genomics of Epithelial Ovarian Cancer: Is Impact on the Management of the Disease, Keynote Speaker, International Symposium on Cancer Research at Mackay Memorial Hospital, Taipei, Taiwan, April 25, 2015

Ovarian Cancer State of the Science, Immunogen, Waltham, MA, May 7, 2015

Retrospective analysis of candidate predictive tumor biomarkers for efficacy in the GOG-0218 trial evaluating front-line carboplatin–paclitaxel with or without bevacizumab for epithelial ovarian cancer, Roche Global Ovarian Cancer Advisory Board Meeting, Zurich Switzerland, May 11, 2015

DFHCC Ovarian Cancer Retreat, Co-Chair, Boston, MA, May 13, 2015

State-of-the-Science - Updates on the Molecular Genetics of Ovarian Cancer and Implications on Management. Edcuational Concepts Group, Satellite Symposium "Optimizing BRCA-Related Ovarian Cancer Treatment: Progress toward Personalizaed Therapy, ASCO Annual Meeting, Chicago, IL, May 29, 2015

Retrospective analysis of candidate predictive tumor biomarkers (BMs) for efficacy in the GOG-

0218 trial evaluating front-line carboplatin-paclitaxel (CP) ± bevacizumab (BEV) for epithelial ovarian cancer (EOC). ASCO Annual Meeting, Chicago, IL, June 1, 2015

Tumor Biology Co-Chair, Oral Abstract Session, ASCO Annual Meeting, Chicago, IL, June 1, 2015

Ovarian Cancer: The State of the Science, Grand Rounds, The Ohio State Univeristy Medical Center, Columbus, OH, June 17-18, 2015

OvaCure Innovation Summit 2015, Copenhagen, Denmark, June 22, 2015

Translational Research on Ovarian Cancer and *Biomarkers in Ovarian Cancer*, Japanese Society of Medical Oncology 2015, 13th Annual Scientific Meeting (JSMO2015), Sapporo, Japan, July 16-17, 2015

The Future Role of Biologic Predictive Factors in the Management Strategy, Ovarian Cancer Workshop, FDA, Silver Spring, MD, September 3, 2015

Clinical Applications of Molecular and Genetic Predictive Factors in Endometrial Cancer, 19th International Meeting of the European Society of Gynaecological Oncology (ESGO 2015), The Acropolis Congress Center, Nice, France, October 24-27, 2015

The Future Role of Biological Markers in the Surgical Treatment Planning, 19th International Meeting of the European Society of Gynaecological Oncology (ESGO 2015), The Acropolis Congress Center, Nice, France, October 24-27, 2015

Future Direction of Ovarian Cancer Research and Clinical Trials, Keynote Lecture, 53rd Annual Meeting of the Japan Society of Clinical Oncology, Kyoto, Japan, October 29-31, 2015

Maximizing the Impact of Antiangiogenesis in Gynecology Oncology, 33rd Chemo Annual Chemotherapy Foundation Symposium: Innovative Cancer Therapy for Tomorrow, New York, NY, November 4-6, 2015

Treatment – Focus on Gynecological Cancers, AACR Cancer Health Disparities Conference, Atlanta, GA, November 13-16, 2015

Identification and characterization of mutations in oncogenes and tumor suppressor genes within cancers of the ovary, endometrial and cervix, AACR Cancer Health Disparities Conference, Atlanta, GA, November 13-16, 2015

Can we predict optimal debulking from the laboratory?, 26th National MITO Meeting, Rome, Italy, December 9-11, 2015

2016 *Integration of biomarkers, correlative, imaging,* NCI, Endometrial Cancer Clinical Trials Planning Meeting, Rockville, Maryland, January 7-8, 2016

GYN Developmental Therapeutics/Phase I/Translational Science Workshop, Chair NRG Oncology Semi-Annual Meeting, Atlanta, GA, January 21-24, 2016

Future of Ovarian Cancer Treatment: Personalized Medicine, Ovarian Cancer Survivors Course, West Palm Beach, FL, February 20, 2016

GOG Foundation/Partners Retreat, Phildelphia, PA, February 26, 2016

Mechanism of Action of PARP Inhibitors and Drug Resistanace, PER Satellite Symposium SGO Expert Perspectives in PARP Inhibition: Evolving Management Strategies in Ovarian Cancer, March 19, 2016

Promising Agents and Strategies in Gynecologic Cancers, Research to Practice Satellite Symposium SGO, March 20, 2016

Biomarkers for Anti-Angiogenic Therapy, Nordic Society of Gynecologic Oncology Roche Symposium and Annual Meeting, Bergen, Norway, April 7, 2016

New Targets in Ovarian Cancer, ANZGOG Annual Scientific Meeting, Sydney Australia, April 14, 2016

Current and future perspectives of the treatment of gynaecological cancer, Platinum Sponsor Breakfast: Roche, ANZGOG Annual Scientific Meeting, Sydney Australia, April 15, 2016

Personalised medicine in gynaecological cancers – how do we get there? ANZGOG Annual Scientific Meeting, Sydney Australia, April 16, 2016

The Molecular Genetics of Epithelial Ovarian Cancer: The Past, Present, and Future Distinguished Clinicians in Oncology Seminar Series, University Hospital of Lausanne (CHUV), Switzerland, April 29, 2016

Molecular Origins of Ovarian Cancer Berlin Institute of Health, Clinical Scientist Summer Symposium on Translational Medicine, July 1-2, 2016

Endometrial Cancer: The Future of Targeted Therapy NRG Oncology Semi-Annual Meeting, Dallas, TX, July 14-17, 2016

DNA Repair & PARP Inhibitors, University of Mississippi CANCER 2016: ASCO and Other Highlights, Jackson, MS, August 19, 2016

The Role of Bevacizumab in Gynecologic Cancers, Roundtable Roche Pharmaceuticals, Vietnam, September 26-30, 2016

Can Patients Be Selected for Anti-Angiogenic Therapy? 16th Biennial Meeting of the International Gynecologic Cancer Society (IGCS), Lisbon, Portugal, October 29, 2016

Current and Emerging Roles for PARP Inhibition in Ovarian Cancer, 34rd Chemo Annual Chemotherapy Foundation Symposium: Innovative Cancer Therapy for Tomorrow, New York, NY, November 9-10, 2016

The Next Giant Leap: Making the Cancer Moonshot a Reality, Elsevier Cancer Research Panel Discussion, Boston, MA, November 16, 2016

GYN update-work targeting BRCA mutations and DNA pathway repair, Molecular and Precision medicine (MAP) Tumor Board Series, Massachusetts General Hospital Cancer Center, Boston, MA, November 28, 2016

Predicting optimal cytoreduction, ISGO, Translation and Innovation, Dublin, Ireland, December 2, 2016

Novel targets in epithelial ovarian cancer, ISGO, Translation and Innovation, Dublin, Ireland, December 3, 2016

2017 *Ovarian Cancer Translational Science Research: Moving the field forward,* Fonadazione IRCCS Istituto Nazionale dei Tumori, Milan, Italy, Janaury 30, 2017

Can biology predict the role of surgery? – The medical point of view. European Institute of Oncology, Milan, Italy, January 31, 2017

Ovarian Cancer: The State of the Science – Instituto di Ricerche Famacologiche Mario Negri, Milan, Italy, February 1, 2017

Ovarian Cancer: The State of the Science, Grand Rounds, The Univeristy of Miami, Miami, FL, February 16-17, 2017

How can molecular abnormalities influence our clinical approach, ESMO Advanced Ovarian Cancer, Valencia, Spain, March 2-3, 2017

Dissecting the Decision: Investigators Discuss Availalbe and Emerging Data Shaping the Management of Common Gynecologic Cancers, Research to Practice Satellite Symposium SGO, March 12, 2017

Ovarian Cancer: The State of the Science, Grand Rounds, Stony Brook School of Medicine, Stony Brook, NY, March 28-29, 2017

Long term survivor in ovarian cancer: what are the codes?, 9th International Charite-Mayo-Conference, Berlin, Germany, May 6, 2017

Ovarian Cancer Risk Reducation and Treatment, 8th Biennial Looking Back Facing Forward, Boston, MA, May 13, 2017

Evolving Evidence: Current Methods to Optimize PARP Inhibition in Multiple Lines of Care, PER

PARP Ovarian Symposium: Redfining Ovarian Cancer Treatment Paradigms by Maximizing Therapeutic Outcomes with PARP Inhibitors, Chicago, IL, June 2, 2017

Presidential Lecture, WAGO 2017 Annual Meeting, Rancho, CA, June 16, 2017

PARP Inhibitors & Beyond: New Developments in the Treatment of Ovarian & Breast Cancer, LEERINK Partners 5th Annual Healthcare Insights Conference, Boston, MA, July 11, 2017

Ovarian Cancer: The State of the Science, Grand Rounds, The University of Kansas Medical Center, Kansas City, MO, October 5, 2017

Proteogenomi Translational Research Centers Session, NIH, The Clinical Proteomic Tumor Analysis Consortium (CPTAC 3.0) Steering Committee Meeting, Bethesda, MA, October 10, 2017

PER Oncology Best Practice Parp Ovarian Program, Per Oncology, St. Louis, MO, December 4, 2017

2018 *Biologic Basis of Ovarian Cancer*, GEMSTONE Meeting, Dallas, TX, February 3, 2018

Which Molecular Markers Have the Potential to Influence Treatment Decisions with PARP Inhibitors? Society of Gynecologic Oncology (SGO) Annual Meeting on Women's Cancer, New Orleans, LA, March 24, 2018.

PARP INHIBITORS: Their Role in the Treatment of Ovarian Cancer, Bio Ascend – St. Mary's Medical Center, Langhorn, PA, March 28, 2018.

Relapsed Ovarian Cancer – Platinum-Sensitive, TRM Oncology EPIC Expert Panel, Chicago, IL, April 28, 2018.

Ovarian Cancer Team, CPTAC PI F2F Meeting, Bethesda, MD, May 2, 2018.

Ovarian Cancer: The State of the Science, UAB Surgery-Pathology-Biomedical Engineering Tri Departmental Seminar Series, Birmingham, AL, May 8, 2018.

Deep South Network, UAB Cancer Control and Population Sciences Program, Birmingham, AL, May 23, 2018.

Physician Education Resources, Evolving Applications for PARP Inhibitors in Ovarian Cancer: Building on a Solid Foundation, American Society of Clinical Oncology (ASCO), Chicago, IL, June 3, 2018.

Ovarian Cancer: The State of the Science, Comprehensive Cancer Center Seminar Series, University of Alabama at Birmingham, September 5, 2018.

State of the Art for Precision Medicine in Gyn Cancers, IGCS 17th Biennial Meeting, Kyoto,

Japan, September 15, 2018.

PTRC: Ovarian Cancer Team, CPTAC PI F2F Meeting, Bethesda, MD, October 16, 2018.

Treatment Approaches and Emerging Therapies and Testing, Pfizer Ovarian Cancer Learning Day, Chicago, IL, November 14-15, 2018.

2019 *Investigator Perspectives on the Current and Future Role of PARP Inhibition in the Management of Ovarian Cancer*, RTP Grand Rounds, Wynnewood, PA, January 31, 2019

GRANT SUPPORT (PAST AND CURRENT)

COMPLETED

PI: Baum 5T32GM7288 Medical Scientist Training Program Role:	1977-1982	$350,000
PI: Torti Five Year Grant from Veterans Administration Department of Defense Role: Co-PI	1988-1993	$495,000
PI: Birrer Intramural Research Award Role: Principal Investigator	1997-1998	$ 30,000
PI: Birrer Intramural Research Award Role: Principal Investigator	1991-2001	$120,000
PI: Boyd CA98027 Director's Challenge Grant Role: Co-PI	2001-2004	$480,000
(Birrer) MGH Proton Program Income (Federal Share)	10/1/2009-9/30/2010	$128,560
PI: Sood OC080465 (W81XH-09OCRP) Early Events in Ovarian Cancer Pathogenesis Role: Principal Investigator for MGH Site	09/01/2009-08/31/2010	$ 13,000
PI: Del Carmen Fidelity Non-Profit Management Foundation	11/1/2009-10/31/2010	$ 50,000

Remote Bold Nan-rod Heating for Ablation of Platinum-resistant Ovarian Cancer Role: Co-PI		
PI: Birrer Osmotic Micro-pump for Delivery of Chemotherapy After Resection of Advanced Ovarian Cancer Role: Principal Investigator	11/1/2009-10/31/2010	$ 50,000
PI: Birrer Phase I/II Study of Carbo & Pralatrexate in Patients with Recurrent Platinum Sensitive Ovarian Cancer, NCCN Role: Principal Investigator	04/01/2010-03/31/11	$300,000
PI: Birrer 3U01CA062490 Dana-Farber Cancer Institute Early Clinical Trials of new Anti-Cancer Agents with Phase I Emphasis Role: Principal Investigator	04/01/2010–05/31/2011	$ 7,874
PI: Birrer 3P50CA105009-0551 Brigham and Women's Hospital, Inc. DF/HCC Ovarian Cancer SPORE – Admin Core Role: Co- Principal Investigator	08/01/2010–07/31/2011	$ 22,599
PI: Godwin Ovarian Cancer Research Foundation, Fox Chase Cancer Center/subaward to MGH Therapeutic Targeting of the Tumor Microenvironment in Ovarian Cancer Role: Principal Investigator	04/01/2009-03/31/2012	$50,000
PI: Birrer 5RC4CA 156551-03 ARRA-NIH-NCI National Cancer Institute Geonomic Stratification of Ovarian Cancer Patients	09/27/2010-8/30/2013	$1,262,809
PI: Birrer KI-DF/HCC 2013-Team 8-0001 Dana-Farber Cancer Institute Title: Osmotic Micro-pump as Delivery System for Intraperitoneal Chemotherapy in the Treatment of Advanced Ovarian Cancer Role: Principal Investigator	03/01/2013-06/30/2015	$309,144
PI: Birrer W81XWH-12-1-0521	09/30/2012-09/29/2015	$200,000

DOD OCRP TLA
Title: Identification of a Genomic Signature Predicting for Recurrence in Early Stage Ovarian Cancer
Role: Principal Investigator

PI: Chabner — 05/15/2012-11/30/2016 — $ 50,412
5P30CA006516-48 (Chabner, Bruce A)
Dana-Farber Cancer Institute
DFHCC - Program Leaders
Role: Co-Investigator

PI: Bast — 09/02/2010-08/31/2015 — $142,000
2P50CA083639-12 The University of Texas MDACC
Title: Early Detection of Epithelial Ovarian Cancer
Role: Principal Investigator for MGH Site

PI: LIU — 04/01/2010-09/30/15 — $ 7,874
NIH CTEP ARRADFHCC Subaward to MGH
Title: A Randomized Trial of Early Palliative Care in Newly Diagnosed Cancer Patients
Role: Principal Investigator for MGH Site

PI: Birrer — 03/22/10 - 01/31/15 — $1,712,199
5R01CA142832-02
NIH-NCI National Cancer Institute
Novel Biomarkers in Ovarian Cancer
Role: Principal Investigator

PI: Disaia — 03/09/09-03/08/16 — $11,067
U10CA27469 Disaia
NIH/NCI/GOG subaward to MGH
Mid-Career Investigator Award
The major goals of this project are to Chair of the Experimental Medicine Committee and Chairman's Advisory Group of the GOG
Role: Principal Investigator

ACTIVE

PI: Birrer — 03/01/2013-02/28/2018 — $282,233
RO1CA169200
NIH-NCI National Cancer Institute
Title: The FGF18/FGFR4 Amplicon: Novel Therapeutic Biomarkers for Ovarian Cancer
Role: Principal Investigator

PI: Skates — 09/24/2010 – 03/31/2021 — $399,746

1U01CA152990-01 (Skates, Steven J)
NIH-NCI National Cancer Institute
Proteomic, Genetic & Longitudinal Pathways to
Ovarian Cancer Biomarker Discovery
Role: Co-Principal Investigator

PI: Birrer 03/28/97 – 03/31/21 $3,524,460
5P30CA013148-45
NIH-NCI National Cancer Institute
Comprehensive Cancer Center Core Support Grant
Role: Principal Investigator

PI: Birrer 01/01/2016 – 12/31/2018 $120,000
128F99-IRG-15-174-56-IRG
American Cancer Society
Institutional Research Grant
Role: Principal Investigator

PI: Birrer 10/01/2015 – 09/30/2017 $300,000
W81XWH-15-1-0139
DOD OCRP
Polymeric RNAI Micorsponge Delivery Simultaneously
Targeting Multiple Genes for Novel Pathway Inhibition of
Ovarian Cancer
Role: Principal Investigator

PI: Birrer 09/01/2016 – 08/31/2018 $250,000
W81XWH-16-1-0593
DOD OCRP-PA
Identification of Novel Ovarian Cancer Oncogenes that
Function by Regulating Exosome Function
Role: Principal Investigator

PI: Birrer 09/01/2016 – 08/31/2019 $1,000,000
W81XWH-16-2-0038
DOD OCRP – Outcomes Consortium Award
The Genomic, Epigenomic, and Quality-of-Life Characteristics
of Long-Term Survivors of Ovarian Cancer
Role: Principal Investigator

PI: Birrer 04/01/2016 – 09/30/2017 $79,209
NCCN
Mechanisms of Sensitivity and Resistance to Mirvetuximab
Soravtansine: Ovarian Cancer and Mesothelioma
Role: Principal Investigator

PI: Paulovich 04/01/2017 – 03/31/2022 $973,291

1U01CA214114-01 NIH-NCI National Cancer Institute Proteogenomic Studies Aimed at Understanding Ovarian Tumor Responses to Agents Targeting the DNA Damage			
PI: Birrer W81XWH-17-1-0225 DOD OCRP Treatment of Recurrent, Platinum-Resistant Ovarian Cancer with Glutaminase 1 and PARP Inhibitors	10/01/2017 – 09/30/2020		$597,663

OTHER (CLINICAL TRIALS)

09-285 A Phase II, Multi-Center, Single-Arm Study Evaluating Carboplatin/Gemcitabine in Combination with BSI-201 for Platinum-Sensitive Recurrent Ovarian Cancer	11/05/2009	PI	$501,504.36
09-286 A Phase II, Multi-Center, Single-Arm Study Evaluating Carboplatin/Gemcitabine in Combination with BSI-201 for Platinum-Resistant Recurrent Ovarian Cancer	11/05/2009	PI	$567,802.90
09-397 The Safety and Efficacy of Combination Therapy With AZD2171 and Temsirolimus in Patients with Recurrent Gynecological Malignancies	01/07/2010	Site PI	$21,018.00
10-113 Phase II, Study of Carboplatin and Pralatrexate in Patients with Recurrent Platinum Sensitive Ovarian, Fallopian Tube or Peritoneal Cancer	02/26/2010	Co PI	$61,829.00
10-258 A Phase II, 2-Stage, 2-Arm PIK3CA Mutation Stratified Trial of MK-2206 in Recurrent or Advanced Endometrial Cancer	03/01/2011	Site PI	$23,074.00
11-057 A Phase I, Open-Label, Dose-Escalation Study of the Safety and Pharmacokinetics of DMUC5754A Administered Intravenously to Patients with Platinum-Resistant Ovarian Cancer	05/30/2012	Site PI	$92,529.74
11-228 A Phase II, Multi-Center, Double-Blind, Placebo Controlled, Randomized Study of Ombrabulin in Patients With Platinum-Sensitive Recurrent Ovarian Cancer Treated With Carboplatin/Paclitaxel	07/28/2011	PI	$53,925.50
11-399 A Randomized Phase II, Non-Comparative Study of The Efficacy of PF-04691502 and PF-05212384 in Patients With Recurrent Endometrial Cancer	02/02/2012	PI	$6,500.00

12-048 A Phase I/II, Open-label, Safety, Pharmacokinetic and Preliminary Efficacy Study of Oral Rucaparib in Patients with gBRCA Mutation Breast Cancer or Other Solid Tumor (CO-338-010)	03/20/2014	Co PI	$54,593.75
12-077 A Phase 1/1b, Multicenter Open-Label, Dose-Escalation and Expansion Study to Evaluate the Safety and Antitumor Activity of MEDI3617, a Human Monoclonal Antibody Directed Against ANG2, as a Single-Agent or in Combination Therapy in Adult Subjects with Advanced Solid Tumors	06/20/2012	PI	$135,913.00
12-159 A Phase I, Study of the Oral PI3kinase Inhibitor BKM120 and the Oral PARP Inhibitor Olaparib in patients with Recurrent Triple Negative Breast Cancer or High Grade Serous Ovarian Cancer	09/01/2012	Site PI	$81,249.00
12-292 A Phase II, Safety and Efficacy Study of Ipilimumab Monotherapy Following Completion of Chemotherapy in Recurrent Platinum Sensitive Ovarian Cancer Subjects with Residual Measurable Disease	09/17/2013	Site PI	$41,230.00
12-312 A Phase I, First-in-Human Study to Evaluate the Safety, Tolerability, Pharmacokinetics of IMGN853 in Adults with Ovarian Cancer and other FOLR1-Positive Solid Tumors	10/01/2012	PI	$127,255.05
13-026 A Randomized, Controlled, Open-Label, Phase II Trial of SGI-110 and Carboplatin in Subjects with Platinum-Resistant Recurrent Ovarian Cancer	05/09/2013	Site PI	$ 88,759.63
13-072 A Phase I, Multiple-Dose Study of the Safety and Tolerability of Single Agent REGN421 Administered Every 2 or 3 Weeks in Patients with Advanced Solid Malignancies	02/19/2013	PI	$50,662.00
13-376 A Phase II, Randomized Double Blind Placebo Controlled Trial of Combination of Pimasertib with SAR245409 or of Pimasertib with SAR245409 Placebo in Subjects with Previously Treated Unresectable Low Grade Ovarian Cancer	11/12/2013	PI	$10,125.00
13-447 A Randomized, Open-Label, Multicenter, Phase II Trial Evaluating the Safety and Activity of DNIB0600A Compared To Pegylated Liposomal Doxorubicin Administered Intravenously to Patients with Platinum-Resistant Ovarian Cancer (GO28609)	07/09/2013	Site PI	$78,912.00
13-491 A Phase Ib, Open-Label, Non-randomized Multicenter	11/20/2013	PI	$12,750.00

Study of Birinapant in Combination with Conatumumab in Subjects with Relapsed Epithelial Ovarian Cancer, Primary Peritoneal Cancer or Fallopian Tube Cancer			
14-050 A Multicenter, Randomized, Double-Blind, Placebo-Controlled Phase III Study of Rucaparib as Switch Maintenance Following Platinum-Based Chemotherapy in Patients with Platinum-Sensitive, High-Grade Serous or Endometoid Epithelial Ovarian, Primary Peritoneal or Fallopian Cancer	07/22/2014	PI	$177,125.00
14-263 Phase I, Open-Label, Dose Escalation, Study of the Safety, Tolerability and Pharmacokinetics of DMUC4064A Administered Intravenously to Patients with Platinum Resistant Ovarian Cancer or Unresectable Pancreatic Cancer (GO29213)	07/18/2014	PI	$650,490.00

OTHER (PATENTS)

Inventor	Case #2026-4120 - Dominant-negative deletion mutants of c-Jun and their use in the prevention and treatment of cancer
Inventor	HHS Ref. no. E-095-2007 – Pro-angiogenic genes in ovarian tumor endothelial cell Isolates
Inventor	HHS Ref. No. E-061-2007/0-PCT-02 - Gene expression profile for predicts ovarian cancer patient survival
Inventor	HHS Ref. No. E-060-2007/0-US-01 - A Gene expression profile that predicts ovarian cancer patient response to chemotherapy
Inventor	USA Serial No. 61/803,919, File March 21, 2013 – Methods and systems for treatment of ovarian cancer
Inventor	USA Serial No. 61/644497, Filed May 9, 2012 - Method and drug delivery device for treatment of ovarian cancer

BIBLIOGRAPHY

MANUSCRIPTS

1. Ju G, **Birrer MJ**, Udem S, and Bloom BR. Complementation analysis of measles virus mutants isolated from persistently infected lymphoblast cell lines. J. Virol. 33:1004-1012, 1980.

2. **Birrer MJ**, Bloom BR, and Udem S. Characterization of measles polypeptides by monoclonal antibodies. Virology 108: 381-390, 1981.

3. **Birrer MJ**, Udem S, Nathenson S, and Bloom BR. Antigenic variants of measles virus. Nature 293: 67-69, 1981.

4. Minna JD, Battey JF, **Birrer MJ**, Brooks BJ, Cuttitta F, DeGreve J, Gazdar AF, Johnson BE, Nau MM, Sausville EA, Seifter E, and Vinocour M. Chromosomal deletion, gene amplification, alternative processing, and autocrine growth factor production in the pathogenesis of human lung cancer. Princess Takamatsu Symp. 17: 109-122, 1986.

5. **Birrer MJ** and Young RC. Differential diagnosis of jaundice in lymphoma patients. Semin. Liver Dis. 7: 269-277, 1987.

6. Kaye F, Battey J, Nau M, Brooks B, Seifter E, DeGreve J, **Birrer MJ**, Sausville E, and Minna, J. Structure and expression of human L-myc gene reveal a complex pattern of alterative mRNA processing. Mol. Cell. Biol. 8:186-195, 1988.

7. Krystal G, **Birrer MJ**, Way J, Nau M, Sausville E, Thompson C, Minna J, and Battey J. Multiple mechanisms for transcriptional regulation of the myc gene family in small-cell lung cancer. Mol. Cell. Biol. 8: 3373-3381, 1988.

8. **Birrer MJ**, Segal S, DeGreve JS, Kaye F, Sausville EA, and Minna JD. L-myc cooperates with ras to transform primary rat embryo fibroblasts. Mol. Cell. Biol. 8:2668-2673, 1988.

9. DeGreve J, Battey J, Fedorko J, **Birrer MJ**, Eva G, Kaye F, Sausville E, and Minna J. The human L-myc gene encodes multiple nuclear phosphoproteins from alternatively processed mRNAs. Mol. Cell. Biol. 8:4381-4388, 1988.

10. **Birrer MJ** and Minna JD. Molecular genetics of lung cancer. Semin. Oncol. 15: 226-235, 1988.

11. Schutte J, Minna JD, and **Birrer MJ**. Deregulated expression of human c-jun transforms primary rat embryo cells in cooperation with an activated c-Ha-ras gene and transforms Rat-1a cells as a single gene. Proc. Natl. Acad. Sci. USA 86:2257-2261, 1989.

12. **Birrer MJ**, Raveh L, Dosaka H, and Segal S. A transfected L-myc gene can substitute for c-myc in blocking murine erythroleukemia cell differentiation. Mol. Cell. Biol. 9:2734-2737, 1989.

13. Takahashi T, Nau MM, Chib I, **Birrer MJ**, Rosenberg RK, Vinocour M, Levitt M, Pass H, Gazdar AF, and Minna, JD. p53: A frequent target for genetic abnormalities in lung cancer. Science 246:491-494, 1989.

14. Minna JD, Schutte J, Viallet J, Thomas F, Kaye FJ, Takahashi T, Nau M, Whang-Peng J, **Birrer MJ**, and Gazdar AF. Transcription factors and recessive oncogenes in the pathogenesis of human lung cancer. Int. J. Cancer 4 (Suppl.): 32-34, 1989.

15. **Birrer MJ** and Minna JD. Genetic changes in the pathogenesis of lung cancer. Annu. Rev. Med. 40: 305-317, 1989.

16. Bliss DP, Jr, Battey JF, Linnoila RI, **Birrer MJ**, Gazdar AF, and Johnson BE. Expression of the atrial natriuretic factor gene in small cell lung cancer tumors and tumor cell lines. J. Natl. Cancer Inst. 82: 305-310, 1990.

17. Dosaka-Akita H, Rosenberg RK, Minna JD, and **Birrer MJ**: A complex pattern of translational initiation and phosphorylation in L-myc proteins. Oncogene 6:371-378, 1991.

18. Kim JH, Takahashi T, Chiba I, Park JG, Birrer MJ, Roh JK, Lee HD, Kim J, Minna JD, and Gazdar AF. Occurrence of p53 gene abnormalities in gastric carcinoma tumors and cell lines. J. Natl. Cancer Inst. 83:938-943, 1991.

19. Szabo E, Preis LH, Brown PH and **Birrer MJ**: The role of jun and fos gene family members in 12-0-tetradecanoylphorbol-13-acetate induced hemopoietic differentiation. Cell Growth Differ. 2:475-482, 1991.

20. Smeal T, Binetruy B, Mercola DA, **Birrer MJ**, and Karin M. Oncogenic and transcriptional cooperation with Ha-Ras requires phosphorylation of c-Jun on serines 63 and 73. Nature 354:494-496, 1991.

21. Alani R, Brown P, Binetruy B, Dosaka H, Rosenberg RK, Angel P, Karin, M, and **Birrer MJ**. The transactivating domain of the c-jun proto-oncoprotein is required for cotransformation of rat embryo cells. Mol. Cell. Biol 11:6286-6295, 1991.

22. Mulshine JL, Linnoila RI, Treston AM, Scott FM, Quinn K, Avis I, Shaw GL, Jensen SM, Brown P, **Birrer MJ**, and Cuttitta F. Candidate biomarkers for application as intermediate end points of lung carcinogenesis. J Cell Biochem16G (Suppl.): 183-186, 1992.

23. **Birrer MJ**, Alani R, Cuttitta F, Preis LH, Sabichi AL, Sanders DA, Siegfried JM, Szabo E, and Brown PH. Early events in the neoplastic transformation of respiratory epithelium. J Natl Cancer Inst Monogr 13:31-38, 1992.

24. Mulshine JL, Linnoila RI, Jensen SM, Magnani JL, Tockman MS, Gupta PK, Scott F S, Avis I, Quinn K, **Birrer MJ** and Cuttitta F. Rational targets for the early detection of lung cancer. . Natl Cancer Inst Monogr 13:183-190, 1992.

25. Mulshine JL, **Birrer MJ**, Treston AM, Scott F, Quinn K, Avis, I, and Cuttitta F. Growth factors and other targets for rational application as intervention agents. Adv Exp Med Biol 320:81-88, 1992.

26. **Birrer MJ** and Brown PH. Application of molecular genetics to the early diagnosis and screening of lung cancer. Cancer Res 52:2658s-2664s, 1992.

27. Bar-Ner M, Messing LT, Cultraro CM, **Birrer MJ**, and Segal S. Regions within the c- myc protein that are necessary for transformation are also required for inhibition of differentation of murine erythroleukemia cells. Cell Growth Differ. 3:183-190, 1992.

28. Barrett J, **Birrer MJ**, Kato GJ, Dosaka-Akita H and Dang CV. Activation domains of L-myc and c-

myc determine their transforming potencies in rat embryo cells. Mol. Cell. Biol. 12:3130-3137, 1992.

29. Brown PB, Alani R, Preis LH, Szabo E, and **Birrer MJ**. Suppression of oncogene- induced transformation by a deletion mutant of c-jun. Oncogene 8:877-886, 1993.

30. Teneriello MG, Ebina M, Linnoila RI, Henry M, Nash JD, Park RC, and **Birrer MJ**. p53 and Ki-ras gene mutations in epithelial ovarian neoplasms. Cancer Res. 53:3103-3108, 1993.

31. Mulshine JL, Treston AM, Brown PH, **Birrer MJ**, and Shaw GL. Initiators and promoters of lung cancer. Chest 103(1 Suppl) 4S-11S, 1993.

32. Sabichi AL and **Birrer MJ**: The molecular biology of lung cancer: application to early detection and prevention. Oncology (Hunting) 7:19-26; discussion 31-32, 37, 1993.

33. Kurie JM, Brown P, Salk E, Scheinberg D, **Birrer MJ**, Deutsch P, and Dmitrovsky E. Cooperation between retinoic acid and phorbol esters enhances human teratocarcinoma differentiation. Differentiation 54:115-122, 1993.

34. Szabo E, **Birrer MJ**, and Mulshine JL. Early detection of lung cancer. Semin. Oncol. 20:374-382, 1993.

35. Brown PH, Chen TK, and **Birrer MJ**. Mechanism of action of a dominant-negative mutant of c-Jun. Oncogene 9:791-799, 1994.

36. Domann FE, Levy JP, **Birrer MJ**, and Bowden GT. Stable expression of a c-JUN deletion mutant in two malignant mouse epidermal cell lines blocks tumor formation in nude mice. Cell Growth Differ. 5:9-16, 1994.

37. Dong Z, **Birrer MJ**, Watts RG, Matrisian LM, and Colburn NH. Blocking of tumor promoter-induced AP-1 activity inhibits induced transformation in JB6 mouse epidermal cells. Proc. Natl. Acad. Sci. USA 91:609-613, 1994.

38. Petrak D, Memon SA, **Birrer MJ**, Ashwell JD, and Zacharchuk CM. A dominant negative mutant of c-jun inhibits NF-AT transcriptional activity and prevents IL-2 gene transcription. J. Immunol. 153:2046-2051, 1994.

39. Rapp UR, Troppmairi J, Beck T, and **Birrer MJ**. Transformation by raf and otheroncogenes renders cells differentially sensitive to growth inhibition by a dominant negative c-jun mutant. Oncogene 9:3493-3498, 1994.

40. Taylor RR, Teneriello MG, Nash JD, Park RC, and **Birrer M**. The molecular genetics of gyn malignancies. Oncology (Huntingt) 8:63-70, 73; discussion 73,78-82, 1994.

41. Szabo E, Preis LH, and **Birrer MJ**. Constitutive cJun expression induces partial macrophage differentiation in U-937 cells. Cell Growth Differ. 5:439-446, 1994.

42. Taylor RR, Linnoila RI, Gerardts J, Teneriello MG, Nash JD, Park RC, and **Birrer MJ**. Abnormal expression of the retinoblastoma gene in ovarian neoplasms and correlation to p53 and K-ras mutations. Gynecol. Oncol. 58:307-311, 1995.

43. Draoui M, Chung P, Park M, **Birrer MJ**, Jakowlew S, and Moody TW. Bombesin stimulates c-fos and c-jun mRNAs in small cell lung cancer cells. Peptides 16:289-292, 1995.

44. Watts RG, Ben-Ari ET, Bernstein LR, **Birrer MJ**, Winterstein D, Wendel E, and Colburn NH. c-jun and multistage carcinogenesis: association of overexpression of introduced c-jun with progression toward a neoplastic endpoint in mouse JB6 cells sensitive to tumor promoter-induced transformation. Mol. Carcinog. 13:27-36, 1995.

45. Cippitelli M, Sica A, Viggiano V, Ye J, Ghosh P, **Birrer MJ**, and Young HA. Negative transcritptional regulation of the interferon-gamma promoter by glucocorticoids and dominant negative mutants of c-Jun. J. Biol. Chem. 270:12548-12556, 1995.

46. Lengye E, Singh B, Gum R, Nerlov C, Sabichi A, **Birrer MJ**, and Boyd D. Regulation of urokinase-type plasminogen activator expression by the v-mos oncogene. Oncogene 11:2639-2648, 1995.

47. **Birrer MJ**. Translational research and epithelial carcinogenesis: molecular diagnostic assays now--molecular screening assays soon? [editorial; comment]. J. Natl. Cancer Inst. 87:1041-1043, 1995.

48. Kim S, Brown PH, and **Birrer MJ**. The inhibitory activity of a transdominant c-jun mutant fused to the ligand binding domain of the estrogen receptor. Oncogene 12:1043-1053, 1996.

49. Freemerman AJ, Turner AJ, **Birrer MJ**, Szabo E, Valerie K, and Grant S. Role of c-jun in human myeloid leukemia cell apoptosis induced by pharmacological inhibitors of protein kinase C. Mol. Pharmacol. 49:788-795, 1996.

50. Antinore MJ, **Birrer MJ**, Patel D, Nader L, and McCance DJ. The human papillomavirus type 16 E7 gene product interacts with and trans-activates the AP1 family of transcription factors. Embo J. 15:1950-1960, 1996.

51. Chen TK, Smith LM, Gebhardt DK, **Birrer MJ**, and Brown PH. Activation and inhibition of the AP-1 complex in human breast cancer cells. Mol. Carcinog. 15:215-226, 1996.

52. Verheij M, Bose R, Lin XH, Yao B, Jarvis WD, Grant S, **Birrer MJ**, Szabo E, Zon LI, Kyriakis JM, Haimovitz-Friedman A, Fuks Z, and Kolesnick RN. Requirement for ceramide-initiated SAPK/JNK signalling in stress-induced apoptosis. Nature 380:75-79, 1996.

53. Szabo E, Riffe ME, Steinberg SM, **Birrer MJ**, and Linnoila RI. Altered cJUN expression: an early event in human lung carcinogenesis. Cancer Res. 56:305-315, 1996.

54. Zia H, Hida T, Jakowlew S, **Birrer MJ**, Gozes Y, Reubi JC, Fridkin M, Gozes I, and Moody TW.

Breast cancer growth is inhibited by vasoactive intestinal peptide (VIP) hybrid, a synthetic VIP receptor antagonist. Cancer Res. 56:3486-3489, 1996.

55. Draoui M, Hida T, Jakowlew S, **Birrer MJ**, Zia F, and Moody TW. PACAP stimulates c-fos mRNAs in small cell lung cancer cells. Life Sci. 59:307-313, 1996.

56. Brown PH, Kim SH, Wise SC, Sabichi AL, and **Birrer MJ**. Dominant-negativemutants of cJun inhibit AP-1 activity through multiple mechanisms and with different potencies. Cell Growth Differ. 7:1013-1021, 1996.

57. Smith LM and **Birrer MJ**. Use of transcription factors as agents and targets for drug development. Oncology (Huntingt) 10:1532-1538; discussion 1541-1542, 1996.

58. Grant S, Freemerman AJ, **Birrer MJ**, Martin AH, Turner AJ, Szabo E, Chelliah J, and Jarvis WD. Effect of 1-_-D-arabinofuranosylcytosine on apoptosis and differentiation in human monocytic leukemia cells (U937) expressing a c-Jun dominant-negative mutant protein (TAM67). Cell Growth Differ. 7:603-613, 1996.

59. Sabichi AL and **Birrer MJ**. Regulation of nuclear oncogenes expressed in lung cancer cell lines. J. Cell. Biochem. 24 (Suppl.):218-227, 1996.

60. Parker MF, Arroyo GF, Geradts J, Sabichi AL, Park RC, Taylor RR, and **Birrer MJ**. Molecular characterization of adenocarcinoma of the cervix. Gynecol. Oncol. 64:242-251, 1997.

61. Potapova O, Haghighi A, Bost F, Liu C, **Birrer MJ**, Gjerset R, and Mercola D. The Jun kinase/stress-activated protein kinase pathway functions to regulate DNA repair and inhibition of the pathway sensitizes tumor cells to cisplatin. J. Biol. Chem. 72:14041-14044, 1997.

62. Hendricks DT, Taylor R, Reed M, and **Birrer MJ**. FHIT gene expression in human ovarian, endometrial, and cervical cell lines. Cancer Res.57:2112-2115, 1997.

63. Smith LM, **Birrer MJ**, Stampfer MR, and Brown PH. Breast cancer cells have lower activating protein 1 transcription factor activity than normal mammary epithelial cells. Cancer Res. 57:3046-3054, 1997.

64. Pisegna JR, Leyton J, Coelho T, Hida T, Jakowlew S, **Birrer MJ**. Fridkin, M, Gozes, I, and Moody, TW. PACAP hybrid: a new PACAP receptor antagonist. Life Sci. 61:631-639, 1997.

65. Zhao B, Yu W, Qian M, Simmons-Menchaca M, Brown P, **Birrer MJ**, Sanders BG, and Kline K. Involvement of activator protein-1 (AP-1) in induction of apoptosis by vitamin E succinate in human breast cancer cells. Mol. Carcinog. 19:180-90, 1997.

66. **Birrer MJ**. Discoveries in the cell cycle and ovarian cancer [editorial]. Gynecol. Oncol. 64:187-188, 1997.

67. Jarvis WD, Fornari FA Jr, Auer KL, Freemerman AJ, Szabo E, **Birrer MJ**, Johnson CR, Barbour

SE, Dent P, and Grant S. Coordinate regulation of stress-and mitogen-activated protein kinases in the apoptotic actions of ceramide and sphingosine. Mol. Pharmacol. 52:935-947, 1997.

68. Hall KL, Teneriello MG, Taylor RR, Lemon S, Ebina M, Linnoila RI, Norris JH, Park RC, and **Birrer MJ**. Anaylsis of Ki-ras, p53, and MDM2 genes in uterine leiomyomas and leiomyosarcomas. Gynecol. Oncol. 65:330-335, 1997.

69. Szabo E, Francis J, and **Birrer MJ**. Alterations in differentiation and apoptosis induced by bufalin in cJun overexpressing U-937 cells. Int. J. Oncol. 12:403-409, 1998.

70. Zagariya A, Mungre S, Lovis R, **Birrer MJ**, Ness S, Thimmapaya B, and Pope R. Tumor necrosis factor alpha gene regulation: enhancement of C/EBP beta-induced activation by c-Jun. Mol. Cell. Biol. 18:2815-2824, 1998.

71. Carter S, Auer KL, Reardon DB, **Birrer MJ**, Fisher PB, Valerie K, Schmidt-Ulrich R, Michelson R, and Dent P. Inhibition of the mutagen activated protein (MAP) kinase cascade potentiates cell killing by low dose ionizing radiation in A431 human squalors carcinoma cells. Oncogene 16:2787-96, 1998

72. Wise SC, Burmeister LA, Zhou XF, Bubulya A, Oberfield JL, **Birrer MJ**, Shemshedini L. Identification of domains of c-Jun mediating androgen receptor transactivation. Oncogene Apr 16;16(15):2001-199.

73. Jacobs-Helber SM, Wickrema A, **Birrer MJ**. Sawyer ST AP1 regulation of proliferation and initiation of apoptosis in erythropoietin-dependent erythroid cells. Mol Cell Biol Jul;18(7):3699-707, 1998 Auer, K.L., 74.

74. Contessa J, Brenz-Verca S, Pirola L, Rusconi S, Cooper G, Abo A, Davis RJ, Wymann MP, **Birrer MJ** and Dent P. Stimulation of DNA synthesis in primary cultures of rat hepatocytes by multiple agonists via a Ras/Rac-Cdc42/SEK/JNK/c-Jun dependent mechanism. Mol. Cell. Biol. 9:561-73, 1998.

75. Sabichi AL, Hendricks DT, Bober MA, and **Birrer MJ**. Retinoic acid receptor expression and growth inhibition of gynecologic cancer cells by the synthetic retinoid N-(4- hydroxyphenyl) retinamide. J. Natl. Cancer Inst. 90:597-605, 1998.

76. Leyton J, Coelho T, Coy DH, Jakowlew S, **Birrer MJ**, Moody TW. PACAP(6-38) inhibits the growth of prostate cancer cells. Cancer Lett 1998 Mar 13;125(1-2):131-9.

77. Yu W, Simmons-Menchaca M, You H, Brown P, **Birrer MJ**, Sanders BG, and Kline K. RRR - tocopheryl succinate induces prolonged activation of c-jun amino-terminal kinase (JNK) and c-jun induction of apoptosis in human MDA-MB-435 breast cancer cells. Mol. Carcinog. 22:247-57, 1998.

78. Dent P, Jarvis WD, **Birrer MJ**, Fisher PB, Schmidt-Ulrich RK, Grant S. The roles of signaling by the p42/p44 mutagen-activated protein (MAP) kinase pathway; a potential route to radio- and

chemo-sensitization of tumor cells resulting in the induction of apoptosis and loss of clonogenicity: Leukemia 1998 Dec;12(12):1843-50.

79. Rao GN, Katki KA, Madamanchi NR, Wu Y, **Birrer MJ**. JunB forms the majority of the AP-1 complex and is a target for redox regulation by receptor tyrosine kinase and G protein-coupled receptor agonists smooth muscle cells. J Biol Chem. 1999 Feb 26; 274(9):6003-10.

80. Geradts J, Maynard R, **Birrer MJ**, Hendricks D, Abbondanzo SL, Fong KM, Barrett JC, Lombardi DP. Frequent loss of KAI1 expression in squamous and lymphoid neoplasms. An immunohistochemical study of archival tissues. Am J Pathol. 1999 Jun;154(6):1665-71.

81. Wang N, Verna L, Hardy S, Zhu Y, Ma KS, **Birrer MJ**, Stermerman MB. c-Jun triggers apoptosis in human endothelial cells. Circ Res. 1999 Sep 3;85(5):387-93.

82. **Birrer MJ**, Hendricks D, Farley J, Sundborg MJ, Bonome T, Walts MJ, Geradts J. Abnormal Fhit expression in malignant and premalignant lesions of the cervix. Cancer Res. 1999 Oct 15;59(20):5270-4.

83. Smith LM, Wise SC, Hendricks DT, Sabichi AL, Bos T, Reddy P; Brown PH, **Birrer MJ**. cJun overexpression in MCF-7 breast cancer cells produces a tumorigenic, invasive and hormone resistant phenotype. Oncogene. 1999 Oct 28;18(44):6063-70.

84. Johnson AC, Murphy BA, Matelis CM, Rubinstein Y, Piebenga EC, Akers LM, Neta G, Vinson C, **Birrer MJ**. Activator protein-1 mediates induced but not basal epidermal growth factor receptor gene expression. Mol Med. 2000 Jan;6(1):17-27.

85. Park JS, Boyer S, Mitchell K, Gilfor D, **Birrer MJ**, Darlington G, El Deiry W, Firestone GL, Munger K, Band V, Fisher PB, Dent P. Expression of human papilloma virus E7 protein causes apoptosis and inhibits DNA synthesis in primary hepatocytes via increased expression of p21(Cip-1/WAF1/MDA6). J Biol Chem 2000 Jan 7;275(1):18-28.

86. Cartee L, Vrana JA, Wang Z, Park JS, **Birrer MJ**, Fisher PB, Grant S, Dent P. Inhibition of the mutagen activated protein kinase pathway potentiates radiation-induced cell killing via cell cycle arrest at the G2/M transition and independently of increased signaling by the JNK/c-Jun pathway. Int J Oncol 2000 Feb;16(2):413-22.

87. Liu H, Sidiropoulos P, Song G, Pagliari LJ, **Birrer MJ**, Stein B, Anrather J, Pope RM. TNF-alpha g expression in macrophages: regulation by NF-kappa B is independent of c-Jun or C/EBP beta. J Immunol 2000 Apr 15;164(8):4277-85.

88. Schmitz MJ, Hendricks DT, Farley J, Taylor RR, Geradts J, Rose GS, **Birrer MJ**. p27 and cyclin D1 abnormalities in uterine papillary serous carcinoma. Gynecol Oncol. 2000 Jun;77(3):439-45.

89. Farley J, Gray K, Nycum L, Prentice M, **Birrer MJ**, Jakowlew SB. Endocervical cancer is associated with an increase in the ligands and receptors for transforming growth factor-beta and a contrasting decrease in p27(Kip1). Gynecol Oncol. 2000 Aug;78(2):113-22.

90. Rinehart-Kim J, Johnston M, **Birrer MJ**, Bos T. Alterations in the gene expression profile of MCF-7 breast tumor cells in response to c-Jun. Int J Cancer. 2000 Oct 15;88(2):180-90.

91. Farley JH, Nycum LR, **Birrer MJ**, Park RC, Taylor RR. Age-specific survival of women with endometrioid adenocarcinoma of the uterus. Gynecol Oncol. 2000 Oct;79(1):86-9.

92. Paasinen-Sohns A, Kielosto M, Kaariainen E, Eloranta T, Laine A, Janne OA, **Birrer MJ**, Holtta E. c-Jun activation-dependent tumorigenic transformation induced paradoxically by over expression or block of S-adenosylmethionine decarboxylase. J Cell Biol. 2000 Nov 13;151(4):801-10.

93. Vos MD, Ellis CA, Bell A, **Birrer MJ**, Clark GJ. Ras uses the novel tumor suppressor RASSF1 as an effector to mediate apoptosis. J Biol Chem. 2000 Nov 17;275(46):35669-72.

94. Kim JS, Pirnia F, Choi YH, Nguyen PM, Knepper B, Tsokos M, Schulte TW, **Birrer MJ**, neuroectodermal tumor cell line in association with RB down-regulation and loss of the G1 checkpoint. Oncogene, 2000 Dec 7;19(52):6082-90.

95. Ebina M, Martinez A, **Birrer MJ**, Ilona Linnoila R. In situ detection of unexpected patterns of mutant p53 gene expression in non-small cell lung cancers. Oncogene. 2001 May 3;20(20):2579-86.

96. Nycum LR, Smith LM, Farley JH, Kost ER, Method MW, **Birrer MJ**. The Role of p27 in Endometrial Carcinoma. Gynecol Oncol. 2001 May;81(2):242-6.

97. Calvisi DF, Donninger H, Vos MD, **Birrer MJ**, Gordon L, Leaner V, Clark GJ. NORE1A tumor suppressor candidate modulates p21CIP1 via p53. Cancer Res. 2009 Jun 1;69(11):4629-37.

98. Fan M, Goodwin ME, **Birrer MJ**, Chambers TC. The c-Jun NH(2)-terminal protein kinase/AP-1 pathway is required for efficient apoptosis induced by vinblastine. Cancer Res. 2001 Jun 1;61(11):4450-8.

99. Krivak TC, McBroom JW, Seidman J, Venzon D, Crothers B, MacKoul PJ, Rose GS, Carlson JW, **Birrer MJ**. Abnormal fragile histidine triad (FHIT) expression in advanced cervical carcinoma: a poor prognostic factor. Cancer Res. 2001 Jun 1;61(11):4382-5.

100. Gardner GJ, **Birrer MJ**. Ovarian tumors of low malignant potential: Can molecular biology solve this enigma? J Natl Cancer Inst. 2001 Aug 1;93(15):1122-3.

101. Manzano RG, Montuenga LM, Dayton M, Dent P, Kinoshita I, Vicent S, Gardner GJ, Nguyen P, Choi YH, Trepel J, Auersperg N, **Birrer MJ**. CL100 expression is down-regulated in advanced epithelial ovarian cancer and its re-expression decreases its malignant potential. Oncogene. 2002 Jun 27; 21(28):443547.

102. Bowen C, **Birrer MJ**, Gelmann EP. Retinoblastoma Protein-mediated Apoptosis After gamma - Irradiation J Biol Chem. 2002 Nov 22;277(47):44969-79.

103. Quong J, Eppenberger-Castori S, Moore D 3rd, Scott GK, **Birrer MJ**, Kueng W, Eppenberger U, Benz CC. Age-dependent changes in breast cancer hormone receptors and oxidant stress markers. Breast Cancer Res Treat. 2002 Dec; 76(3):221-36.

104. Farley J, Smith LM, Darcy KM, Sobel E, O'Connor D, Henderson B, Morrison LE, **Birrer MJ**, Gynecologic Oncology Group. Cyclin E expression is a significant predictor of survival in advanced, suboptimally debulked ovarian epithelial cancers: a Gynecologic Oncology Group study. Cancer Res. 2003 Mar 15; 63(6):1235-41.

105. Dent P, Qiao L, Gilfor D, **Birrer MJ,** Grant S, Fisher PB. The regulation of tumor suppressor genes by ocogenes Methods Mol Biol. 2003 222:269-92.

106. Shen YH, Godlewski J, Zhu J, Sathyanarayana P, Leaner V, **Birrer MJ**, Rana A, Tzivion G. Cross-talk between JNK/SAPK and ERK/MAPK pathways: sustained activation of JNK blocks ERK activation by mitogenic factors. J Biol Chem. 2003 278(29):26715-21. Epub 2003 May 08.

107. Kinoshita I, Leaner V, Katabami M, Manzano RG, Dent P, Sabichi A, **Birrer MJ**. Identification of cJun- responsive genes in Rat-1a cells using multiple techniques: increased expression of stathmin is necessary or cJun-mediated anchorage-independent growth. Oncogene. 2003 May 8;22(18):2710-22.

108. Leaner VD, Kinoshita I, **Birrer MJ**. AP-1 complexes containing cJun and JunB cause cellular transformation of Rat1a fibroblasts and share transcriptional targets. Oncogene. 2003 Aug 28;22(36):5619-29.

109. Trepel J, **Birrer MJ**. A Link Between Histone Deacetylase Inhibitors and NF- kappaB Signaling. Cell Cycle. 2003 Sep-Oct; 2(5):452-3. No abstract available.

110. Winter WE, Seidman JD, Krivak TC, Chauhan S, Carlson JW, Rose GS, **Birrer, MJ**. Clinicopathological analysis of c-kit expression in carcinosarcomas and leiomyosarcomas of the uterine corpus. Gynecol Oncol. 2003 Oct;91(1):3-8.

111. Romashko J, Horowitz S, Franek WR, Palaia T, Miller EJ, Lin A, **Birrer MJ**, Scott W, Mantell LL. MAPK pathways mediate hyperoxia-induced oncotic cell death in lung epithelial cells. Free Radic Biol Med. 2003 Oct 15;35(8):978-93.

112. Zorn KK, Jazaeri AA, Awtrey CS, Gardner GJ, Mok SC, Boyd J, **Birrer MJ**. Choice of normal ovarian control influences determination of differentially expressed genes in ovarian cancer expression profiling studies. Clin Cancer Res. 2003 Oct 15; 9(13):4811-8.

113. Farley JH, **Birrer MJ**. Biologic directed therapies in gynecologic oncology. Curr Oncol Rep. 2003 Nov; 5(6):459-67.

114. Zeinoun Z, Teugels E, Vermeij J, Neyns B, **Birrer MJ**, De Greve J. Restoration of an impaired TGF-beta1 autocrine growth-inhibitory circuit results in growth inhibition of ovarian epithelial cancer cells and complete inhibition of their tumorigenicity. Cancer Detect Prev. 2003; 27(5):380-8.

115. Farley J, Uyehara C, Hashiro G, Belnap C, **Birrer MJ**, Salminen E. Cyclooxygenase-2 expression predicts recurrence of cervical dysplasia following loop electrosurgical excision procedure. Gynecol Oncol. 2004 Feb; 92(2):596-602.

116. Liu S, Brown CW, Berlin KD, Dhar A, Guruswamy S, Brown D, Gardner GJ, **Birrer MJ**, Benbrook DM. Synthesis of flexible sulfur-containing heteroarotinoids that induce apoptosis and reactive oxygen species with discrimination between malignant and benign cells. J Med Chem. 2004 Feb 12; 47(4):999-1007.

117. Hommura F, Katabami M, Leaner VD, Donninger H, Sumter TF, Resar LM, **Birrer MJ**. HMG-I/Y is a c-Jun/activator protein-1 target gene and is necessary for c-Jun-induced anchorage-independent growth in Rat1a cells. Mol Cancer Res. 2004 May; 2(5):305-14.

118. Cuello M, Coats AO, Darko I, Ettenberg SA, Gardner GJ, Nau MM, Liu JR, **Birrer MJ**, Lipkowitz S. N-(4-hydroxyphenyl) retinamide (4HPR) enhances TRAIL-mediated apoptosis through enhancement of a mitochondrial-dependent amplification loop in ovarian cancer cell lines. Cell Death Differ. 2004 May; 11(5):527-41.

119. Zou X, Jordan RC, Mok S, **Birrer MJ**, Wong DT. DNA copy number abnormality of oral squamous cell carcinoma detected with cDNA array-based comparative genomic hybridization. Cancer Genet Cytogenet. 2004 May; 151(1):90-2.

120. Kielosto M, Nummela P, Katainen R, Leaner V, **Birrer MJ**, Holtta E: Reversible regulation of the transformed phenotype of ornithine decarboxylase- and ras-overexpressing cells by dominant-negative mutants of c-Jun. Cancer Res. 2004 Jun 1;64(11):3772-9.

121. Sherman, ME, Berman, J., **Birrer, MJ**, Cho KR, Ellenson, LH, Gorstein, F, and Seidman, JD. Current challenges and opportunities for research on borderline ovarian tumors. Human Pathology 2004 Aug; 35(8):961-70.

122. Elagib KE, Xiao M, Hussainin IM, Delehanty LL, Palmer LA, Racke FK, **Birrer MJ**, McDevitt MA, and Goldfarb AN. Jun blockage of erythropoiesis: a role for repression of GATA-1 by HERP2 Mol. Cell. Biol 2004 Sep; 24(17):7779-94.

123. Donninger H, Bonome T, Radonovich M, Pise-Masison CA, Brady J, Shih J, Barrett JC, and **Birrer MJ**. Whole genome expression profiling of advance stage papillary serous ovarian cancer reveals activated pathways. Oncogene 2004 Oct21;23(49):8065-77.

124. Tsuda H, Bandera CA, **Birrer MJ**, Hashiguchi Y, Berkowitz RS, Mok SC. Cyclin E amplification and over expression in clear cell adenocarcinoma of the ovary. Oncology 2004; 67(3-4):291-9.

125. Tsuda H, **Birrer MJ**, Ito YM, Ohashi Y, Lin M,Lee C, Wong WH, Rao PH, Lau CC, Berkowitz RS, Wong KK, Mok SC. Identification of DNA copy number changes in microdissected serous ovarian cancer tissue using a cDNA microarray platform Cancer Genet Cytogenet 2004 Dec;155(2):97-107.

126. Elam C, Hesson L, Vos MD, Eckfeld K, Ellis C, Bell A, Taylor B S, Krex D, **Birrer MJ**, Latif F,

and Clark G. RRP22 is a farnesylated, nucleolar, Ras-related protein with tumor suppressor potential. 2005 Cancer Research. Apr 15; 65(8): 3117-25.

127. Leaner VD, Donninger H, Ellis CA, Clark GJ, and **Birrer MJ**. P75-Ras-GRF1 is a c-Jun/AP-1 target protein: its up regulation results in increased Ras activity and is necessary for c-Jun-induced nonadherent growth of Rat1a cells. Mol Cell Biol. 2005 Apr; 25(8):3324-37.

128. Katabami M, Donninger H, Hommura F, Leaner VD, Kinoshita I, and **Birrer MJ**. Cyclin A is a cJun target gene regulated by direct and indirect mechanisms and is necessary for cJun-induced anchorage independent growth in Rat1a cells. J Biol. Chem 2005 Apr 29; 280(17):16728-38.

129. Jazaeri AA, Awtrey CS, Chandramouli GV, Chuang YE, Khan J, Sotiriou C, Aprelikova O, Yee CJ, Zorn KK, **Birrer MJ**, Barrett JC, Boyd. Gene expression profiles associated with response to chemotherapy in epithelial ovarian cancers. Clin Cancer Res. 2005 Sep 1; 11(17):6300-10.

130. Zorn KK, Bonome T, Gangi L, Chandramouli GV, Awtrey CS, Gardner GJ, Barrett JC, Boyd J, and **MJ Birrer**. Gene expression profiles of serous, endometrioid, and clear cell subtypes of ovarian and endometrial cancer. Clin Cancer Res. 2005 Sep 15;11(18):6422-30.

131. Tsuda H, Ito YM, Ohashi Y, Wong KK, Hashiguchi Y, Welch WR, Berkowitz RS, **Birrer MJ**, and Mok SC. Identification of over expression and amplification of ABCF2 in clear cell ovarian adenocarcinomas by cDNA microarray analyses. Clin Cancer Res. 2005 Oct 1; 11(19 Pt 1):6880-8.

132. Bonome T, Lee JY, Park DC, Radonovich M, Pise-Masison C, Brady J, Gardner GJ, Hao K, Wong WH, Barrertt JC, Lu KH, Sood AK, Gershenson DM, Mok SC, **Birrer, MJ**. Expression profiling of serous low malignant potential, low-grade, and high-grade tumors of the ovary. Cancer Res. 2005 Nov 15; 65(22):10602-12.

133. Nummela P, Yin M, Kielosto M, Leaner V, **Birrer MJ**, Holtta E. Thymosin beta4 is a determinant of the transformed phenotype and invasiveness of S-adenosylmethionine decarboxylase-transfected fibroblasts. Cancer Res. 2006 Jan 15; 66(2):701-12.

134. Wamunyokoli FW, Bonome T, Lee JY, Feltmate CM, Welch WR, Radonovich M, Pise-Masison C, Brady J, Hao K, Berkowitz RS, Mok S, **Birrer MJ**. Expression profiling of mucinous tumors of the ovary identifies genes of clinicopathologic importance. Clin Cancer Res. 2006 Feb 1; 12(3 Pt 1):690-700.

135. DiFeo A, Narla G, Hirshfeld J, Camacho-Vanegas O, Narla J, Rose SL, Kalir T, Yao S, Levine A, **Birrer MJ**, Bonome T, Friedman SL, Buller RE, Martignetti JA. Roles of KLF6 and KLF6-SV1 in ovarian cancer progression and intraperitoneal dissemination. Clin Cancer Res. 2006 Jun 15; 12(12):3730-9.

136. **Birrer MJ**. Ovarian cancer: not a borderline issue! Cancer Biol Ther. 2006 Jul; 5(7):786-7.

137. Sunde JS, Donninger H, Wu K, Johnson ME, Pestell RG, Rose GS, Mok SC, Brady J, Bonome T, **Birrer MJ**. Expression Profiling Identifies Altered Expression of Genes That Contribute to the

Inhibition of Transforming Growth Factor-{beta} Signaling in Ovarian Cancer. Cancer Res. 2006 Sep 1; 66(17):8404-12.

138. Fountain J, Trimble E, **Birrer MJ**. Summary and discussion of session recommendations. Gynecol Oncol. 2006 Nov; 103(2 Suppl. 1):S23-5.

139. Farley J, Bast RC Jr, **Birrer MJ**. Biomarkers and clinical trial design. Gynecol Oncol. 2006 Nov; 3(2 Suppl 1):S3-5.

140. Farley J, **Birrer MJ**, Christian M.C. The future of phase II trials. Gynecol Oncol. 2006 Nov; 103(2 Suppl 1):S20.

141. Fountain J, Trimble EL, **Birrer MJ**. Introduction. Gynecol Oncol. 2006 Nov; 103(2 Suppl 1):S1.

142. Wu K, Liu M, Li A, Donninger H, Rao M, Jiao X, Lisanti MP, Cvekl A, **Birrer MJ**, Pestell RG. The Cell Fate Determination Factor DACH1 Inhibits c-Jun Induced Contact-Independent Growth. Mol Biol Cell. 2007 Mar;18(3):755-67.

143. Mok SC, Elias KM, Wong KK, Ho K, Bonome T, **Birrer MJ**. Biomarker discovery in epithelial ovarian cancer by genomic approaches. Adv Cancer Res. 2007; 96:1-22.

144. Lu C, Bonome T, Li Y, Kamat AA, Han LY, Schmandt R, Coleman RL, Gershenson DM, Jaffe RB, **Birrer MJ**, Sood AK. Gene alterations identified by expression profiling in tumor-associated endothelial cells from invasive ovarian carcinoma. Cancer Res. 2007 Feb 15; 67(4):1757-68.

145. Wong YF, Cheung TH, Lo KW, Yim SF, Siu NS, Chan SC, Ho TW, Wong KW, Yu MY, Wang VW, Li C, Gardner GJ, Bonome T, Johnson WB, Smith DI, Chung TK, and **Birrer MJ**. Identification of molecular markers and signaling pathway in endometrial cancer in Hong Kong Chinese women by genome-wide gene expression profiling. Oncogene. 2007 Mar 22; 26(13):1971-82.

146. Gershenson DM, **Birrer MJ**. Time for action: a "sea change" in treatment strategies for rare types of epithelial ovarian cancer. Gynecol Oncol. 2007 Jul; 106(1):1-3.

147. **Birrer MJ**, Johnson ME, Hao K, Wong KK, Park DC, Bell A, Welch WR, Berkowitz RS, Mok SC. Whole genome oligonucleotide-based array comparative genomic hybridization analysis identified fibroblast growth factor 1 as a prognostic marker for advanced-stage serous ovarian adenocarcinomas. J Clin Oncol. 2007 Jun 1; 25(16):2281-7. Erratum in: J Clin Oncol. 2007 Jul 20; 25(21):3184.

148. Kajanne R, Miettinen P, Mehlem A, Leivonen SK, **Birrer MJ**, Foschi M, Kahari VM, Leppa S. EGF-R regulates MMP function in fibroblasts through MAPK and AP-1 pathways. J Cell Physiol. 2007 Aug; 212(2):489-97.

149. Allen NP, Donninger H, Vos MD, Eckfeld K, Hesson L, Gordon L, **Birrer MJ**, Latif F, Clark GJ. RASSF6 is a novel member of the RASSF family of tumor suppressors. Oncogene 2007 Sep

13;26(42):6203-11.

150. Kelloff GJ, Sullivan DC, Baker H, Clarke LP, Nordstrom R, Tatum JL, Dorfman GS, Jacobs P, Berg CD, Pomper MG, **Birrer MJ**, Tempero M, Higley HR, Petty BG, Sigman CC, Maley C, Sharma P, Wax A, Ginsberg GG, Dannenberg AJ, Hawk ET, Messing EM, Grossman HB, Harisinghani M, Bigio IJ, Griebel D, Henson DE, Fabian CJ, Ferrara K, Fantini S, Schnall MD, Zujewski JA, Hayes W, Klein EA, DeMarzo A, Ocak I, Ketterling JA, Tempany C, Shtern F, Parnes HL, Gomez J, Srivastava S, Szabo E, Lam S, Seibel EJ, Massion P, McLennan G, Cleary K, Suh R, Burt RW, Pfeiffer RM, Hoffman JM, Roy HK, Wang T, Limburg PJ, El-Deiry WS, Papadimitrakopoulou V, Hittelman WN, MacAulay C, Veltri RW, Solomon D, Jeronimo J, Richards-Kortum R, Johnson KA, Viner JL, Stratton SP, Rajadhyaksha M, Dhawan A: Workshop Program Committee. Workshop on imaging science development for cancer prevention and preemption. Cancer Biomarkers. 2007; 3(1):1-33.

151. Whibley CE, McPhail KL, Keyzers RA, Maritz MF, Leaner VD, **Birrer MJ**, Davies-Coleman MT, Hendricks DT. Reactive oxygen species mediated apoptosis of esophageal cancer cells induced by marine triprenyl toluquinones and oluhydroquinones. Mol Cancer Ther. 2007 Sep;6(9):2535-43.

152. Farley J, Ozbun L, Samimi G, **Birrer MJ**. Cell cycle and related proteins. Dis Markers. 2007;23(5-6):433-43.

153. Samimi G, Ozbun L, Johnson ME, Mok SC, **Birrer MJ**. Biomarkers of mucinous tumors of the ovary. Dis Markers. 2007;23(5-6):389-96.

154. Mok SC, Kwong J, Welch WR, Samimi G, Ozbun L, Bonome T, **Birrer MJ**., Berkowitz RS, Wong KK. Etiology and pathogenesis of epithelial ovarian cancer. Dis Markers. 2007;23 (5-6):367-76.

155. Irarrazabal CE, Williams CK, Ely MA, **Birrer MJ**, Garcia-Perez A, Burg MB, Ferraris JD. AP-1 contributes to high NaCl-induced increase in TonEBP/OREBP transactivating activity. J Biol Chem. 2008 Feb 1;283(5):2554-63.

156. Landen CN Jr, **Birrer MJ**, Sood AK. Early events in the pathogenesis of epithelial ovarian cancer. J Clin Oncol. 2008 Feb 20;26(6):995-1005.

157. Zudaire E, Cuesta N, Murty V, Woodson K, Adams L, Gonzalez N, Martínez A, Narayan G, Kirsch I, Franklin W, Hirsch F, **Birrer MJ**, Cuttitta F. The aryl hydrocarbon receptor repressor is a putative tumor suppressor gene in multiple human cancers. J Clin Invest. 2008 Feb;118(2):640-50.

158. Shimizu Y, Kinoshita I, Kikuchi J, Yamazaki K, Nishimura M, **Birrer MJ**, Dosaka-Akita H. Growth inhibition of non-small cell lung cancer cells by AP-1 blockade using a cJun dominant-negative mutant. Br J Cancer. 2008 Mar 11;98(5):915-22.

159. Litkouhi B, Fleming E, Welch WR, Berkowitz RS, **Birrer MJ,** Mok SC. Overexpression of CEACAM6 in borderline and invasive mucinous ovarian neoplasms. Gynecol Oncol. Gynecol

Oncol. 2008 May;109(2):234-9

160. Zhang L, Volinia S, Bonome T, Calin GA, Greshock J, Yang N, Liu CG, Giannakakis A, Alexiou P, Hasegawa K, Johnstone CN, Megraw MS, Adams S, Lassus H, Huang J, Kaur S, Liang S, Sethupathy P, Leminen A, Simossis VA, Sandaltzopoulos R, Naomoto Y, Katsaros D, Gimotty PA, DeMichele A, Huang Q, Bützow R, Rustgi AK, Weber BL, **Birrer MJ**, Hatzigeorgiou AG, Croce CM, Coukos G. Genomic and epigenetic alterations deregulate microRNA expression in human epithelial ovarian cancer. Proc Natl Acad Sci U S A. 2008 May 13;105(19):7004-9.

161. Farley J, Ozbun LL, **Birrer MJ**. Genomic analysis of epithelial ovarian cancer. Cell Res. 2008 May;18(5):538-48.

162. Stany MP, Bonome T, Wamunyokoli F, Zorn K, Ozbun L, Park DC, Hao K, Boyd J, Sood AK, Gershenson DM, Berkowitz RS, Mok SC, **Birrer MJ**. Classification of ovarian cancer: a genomic analysis. Adv Exp Med Biol. 2008;622:23-33.

163. Bonome T, Levine DA, Shih J, Randonovich M, Pise-Masison CA, Bogomolniy F, Ozbun L, Brady J, Barrett JC, Boyd J, **Birrer MJ**. A gene signature predicting for survival in suboptimally debulked patients with ovarian cancer. Cancer Res. 2008 Jul 1;68(13):5478-86.

164. Sood AK, **Birrer MJ**. Highlights from the 2008 Annual Meeting of the American Association for Cancer Research. Gynecol Oncol. 2008 Jul; 110(1):5-6.

165. Park DC, Yeo SG, Wilson MR, Yerbury JJ, Kwong J, Welch WR, Choi YK, **Birrer MJ**, Mok SC, Wong KK. Clusterin interacts with Paclitaxel and confer Paclitaxel resistance in ovarian Cancer. Neoplasia 2008 Sep; 10(9):964-72169.

166. Lorenzi PL, Llamas J, Gunsior M, Ozbun L, Reinhold WC, Varma S, Ji H, Kim H, Hutchinson AA, Kohn EC, Goldsmith PK, **Birrer MJ**, Weinstein JN. Asparagine synthetase is a predictive biomarker of L-asparaginase activity in ovarian cancer cell lines. Mol Cancer Ther 2008 Oct;7(10):3123-8.

167. Anglesio MS, Arnold JM, George J, Tinker AV, Tothill R, Waddell N, Simms L, Locandro B, Fereday S, Traficante N, Russell P, Sharma R, Birrer MJ; AOCS Study Group, deFazio A, Chenevix-Trench G, Bowtell DD. Mutation of ERBB2 provides a novel alternative mechanism for the ubiquitous ctivation of RAS-MAPK in ovarian serous low malignant potential tumors. Mol Cancer Res. 2008 Nov; 6(11): 1678-90.

168. Kikuchi J, Kinoshita I, Shimizu Y, Oizumi S, Nishimura M, **Birrer MJ**, Dosaka-Akita H. Simultaneous blockade of AP-1 and phosphatidylinositol 3-kinase pathway in non-small cell lung cancer cells. Br J Cancer. 2008 Dec 16;99(12):2013-9.

169. Arora S, Wang Y, Jia Z, Vardar-Sengul S, Munawar A, Doctor KS, **Birrer MJ**, McClelland M, Adamson E, Mercola D. Egr1 regulates the coordinated expression of numerous EGF receptor target genes as identified by ChIP-on-chip. Genome Biol. 2008; 9(11):R166.

170. Callahan MJ, Nagymanyoki Z, Bonome T, Johnson ME, Litkouhi B, Sullivan EH, Hirsch MS, Matulonis UA, Liu J, **Birrer MJ,** Berkowitz RS, Mok SC. Increased HLA-DMB expression in the tumor epithelium is associated with increased CTL infiltration and improved prognosis in advanced-stage serous ovarian cancer. Cancer Res. 2008 Dec 1;14(23):7667-73.

171. Leaner VD, Chick JF, Donninger H, Linniola I, Mendoza A, Khanna C, **Birrer MJ**. Inhibition of AP-1 transcriptional activity blocks the migration, invasion, and experimental metastasis of murine osteosarcoma. Am J Pathol. 2009 Jan;174(1):265-75.

172. Merritt WM, Lin YG, Han LY, Kamat AA, Spannuth WA, Schmandt R, Urbauer D, Pennacchio LA, Cheng JF, Nick AM, Deavers MT, Mourad-Zeidan A, Wang H, Mueller P, Lenburg ME, Gray JW, Mok S, **Birrer MJ**, Lopez-Berestein G, Coleman RL, Bar-Eli M, Sood AK. Dicer, Drosha, and outcomes in patients with ovarian cancer N Engl J Med.
2008 Dec 18;359(25):2641-50.

173. Kitchener HC, Trimble EL, **Birrer MJ**, Endometrial Cancer Working Group of the Gynecologic Cancer Intergroup. Endometrial Cancer State of the Science Meeting. Int J Gynecol Cancer. 2009 Jan;19(1):134-40.

174. Chung TK, Cheung TH, Huen NY, Wong KW, Lo KW, Yim SF, Siu NS, Wong YM, Tsang PT, Pang MW, Yu MY, To KF, Mok SC, Wang VW, Li C, Cheung AY, Doran G, **Birrer MJ**, Smith DI, Wong YF. Dysregulated microRNAs and their predicted targets associated with endometrioid endometrial adenocarcinoma in Hong Kong women.
Int J Cancer 2009 Mar 15; 24(6):1358-65167.

175. Singh S, Manda SM, Sikder D, **Birrer MJ**, Rothermel BA, Garry DJ, Mammen PP. Calcineurin activates cytoglobin transcription in hypoxic myocytes. J Biol Chem. 2009 Apr 17;284(16):10409-21.

176. van der Watt PJ, Maske CP, Hendricks DT, Parker MI, Denny L, Govender D, **Birrer MJ**, Leaner VD. The Karyopherin proteins, Crm1 and Karyopherin beta1, are overexpressed in cervical cancer and are critical for cancer cell survival and proliferation. Int J Cancer. 2009 Apr 15;124(8):1829-40.

177. Wang B, Khachigian LM, Esau L, **Birrer MJ**, Zhao X, Parker MI, Hendricks DT. A key role for early growth response-1 and nuclear factor-kappaB in mediating and maintaining GRO/CXCR2 proliferative signaling in esophageal cancer. Mol Cancer Res. 2009 May;7(5):755-64.

178. Farley J, Fuchiuji S, Darcy KM, Tian C, Hoskins WJ, McGuire WP, Hanjani P, Warshal D, Greer BE, Belinson J, **Birrer MJ**. Associations between ERBB2 amplification and progression-free survival and overall survival in advanced stage, suboptimally-resected epithelial ovarian cancers: a Gynecologic Oncology Group Study. Gynecol Oncol. 2009 Jun;113(3):341-7.

179. Nosov V, Su F, Amneus M, **Birrer MJ,** Robins T, Kotlerman J, Reddy S, Farias-Eisner R. Validation of serum biomarkers for detection of early-stage ovarian cancer. Am J Obstet Gynecol. 2009 Jun;200(6):639.e1-5.

180. Calvisi DF, Donninger H, Vos MD, **Birrer MJ**, Gordon L, Leaner V, Clark GJ. NORE1Atumor suppressor candidate modulates p21CIP1 via p53. Cancer Res.2009 Jun 1;69(11):4629-37. Epub 2009 May 12

181. Kwong J, Chan FL, Wong KK, **Birrer MJ**, Archibald KM, Balkwill FR, Berkowitz RS, Mok SC. Inflammatory cytokine tumor necrosis factor alpha confers precancerous phenotype in an organoid model of normal human ovarian surface epithelial cells. Neoplasia. 2009 Jun;11(6):529-41.

182. Terry KL, Vitonis AF, Hernandez D, Lurie G, Song H, Ramus SJ, Titus-Ernstoff L, Carney ME, Wilkens LR, Gentry-Maharaj A, Menon U, Gayther SA, Pharaoh PD, Goodman MT, Cramer DW, **Birrer MJ**. A polymorphism in the GALNT2 gene and ovarian cancer risk in four population based case-control studies. Int J Mol Epidemiol Genet. 2010 Jul 26;1(4):272-7

183. Demelash A, Kim H, Jensen-Taubman S, Dakir EH, Ozbun L, **Birrer MJ**, Linnoila, RI. Matrilysin-1 Mediates Bronchiolization of Alveoli, A Potential Premalignant Change in Lung Cancer. J Pathol. 2009 Aug;175(2):592-604.

184. Wang XY, Demelash A, Kim H, Jensen-Taubman S, Dakir el H, Ozbun L, **Birrer MJ**, Linnoila RI. Matrilysin-1 mediates bronchiolization of alveoli, a potential premalignant change in lung cancer. Am J Pathol. 2009 Aug;175(2):592-604.

185. Darcy KM, Brady WE, Blancato JK, Dickson RB, Hoskins WJ, McGuire WP, **Birrer MJ**. Prognostic relevance of c-MYC gene amplification and polysomy for chromosome 8 in suboptimally-resected, advanced stage epithelial ovarian cancers: A Gynecologic Oncology Group study. Gynecol Oncol. 2009 Sep;114(3):472-9.

186. Farley JH, Tian C, Rose GS, Brown CL, **Birrer MJ**, Maxwell GL. Race does not impact outcome for advanced ovarian cancer patients treated with cisplatin/paclitaxel: an analysis of Gynecologic Oncology Group trials. Cancer. 2009 Sep 15;115(18):4210-7.

187. Farley J, **Birrer MJ**. Novel Therapeutic Targets, Cancer Treat Res. 2009;149:63-84.

188. Mok SC, Bonome T, Vathipadiekal V, Bell A, Johnson ME, Wong KK, Park DC, Hao K, Yip DK, Donninger H, Ozbun L, Samimi G, Brady J, Randonovich M, Pise-Masison CA, Barrett JC, Wong WH, Welch WR, Berkowitz RS, **Birrer MJ**. A gene signature predictive for outcome in advanced ovarian cancer identifies a survival factor: microfibril- associated glycoprotein 2. Cancer Cell. 2009 Dec 8;16(6):521-32.

189. Na YJ, Farley J, Zeh A, del Carmen M, Penson R, **Birrer MJ**. Ovarian cancer: markers of response. Int J Gynecol Cancer. 2009 Dec;19 Suppl 2:S21-9.

190. Farley JH, Tian C, Rose GS, Brown CL, **Birrer MJ**, Risinger JI, Thigpen JT, Fleming GF, Gallion HH, Maxwell GL. Chemotherapy intensity and toxicity among black and white women with advanced and Recurrent endometrial cancer: A Gynecologic Oncology Group Study. Cancer. 2010;Jan 15;116(2):355-61.

191. Minig L, Trimble EL, **Birrer MJ**, Kim KY, Takebe N, Abrams JS. NIH and NCI support for development of novel therapeutics in gynecologic cancer: a user's guide Gynecol Oncol. 2010 Feb;116(2):177-80.

192. Clauss A, Ng V, Liu J, Piao H, Russo M, Vena N, Sheng Q, Hirsch MS, Bonome T, Matulonis U, Ligon AH, **Birrer MJ**, Drapkin R. Overexpression of elafin in ovarian carcinoma is driven by genomic gains and activation of the nuclear factor kappaB pathway and is associated with poor overall survival. Neoplasia. 2010 Feb;12(2):161-72.

193. Byron SA, Gartside MG, Wellens CL, Goodfellow PJ, **Birrer MJ**, Campbell IG, Pollock PM FGFR2 mutations are rare across histologic subtypes of ovarian cancer. Gynecol Oncol. 2010 Apr;117(1):125-9.

194. Huh WK, Sill MW, Darcy KM, Elias KM, Hoffman JS, Boggess JF, Alvarez RD, Long HJ, O'Malley DM, **Birrer MJ**. Efficacy and safety of imatinib mesylate (Gleevec) and immunohistochemical expression of c-Kit and PDGFR-beta in a Gynecologic Oncology Group Phase Il Trial in women with recurrent or persistent carcinosarcomas of the uterus. Gynecol Oncol. 2010 May;117(2):248-54.

195. Hernandez L, Hsu SC, Davidson B, **Birrer MJ**, Kohn EC, Annunziata CM. Activation of NF-kappaB signaling by inhibitor of NF-kappaB kinase beta increases aggressiveness of ovarian cancer. Cancer Res. 2010 May 15;70(10):4005-14.

196. Darcy KM, **Birrer MJ**. Translational research in the Gynecologic Oncology Group: evaluation of ovarian cancer markers, profiles, and novel therapies. Gynecol Oncol. 2010 Jun;117(3):429-39.

197. Annunziata CM, Stavnes HT, Kleinberg L, Berner A, Hernandez LF, **Birrer MJ**, Steinberg SM, Davidson B, Kohn EC. Nuclear factor kappaB signaling by inhibitor NF-kappa beta increases aggressiveness of ovarian cancer. Cancer. 2010 Jul 1;116(13):3276-84.

198. Annunziata, CM **Birrer MJ**. CD157 in ovarian carcinoma: How does it help us? J Natl Cancer Inst. 2010 Aug 4;102(15):1104-5.

199. Lu C, Han HD, Mangala LS, Ali-Fehmi R, Newton CS, Ozbun L, Armaiz-Pena GN, Hu W, Stone RL, Munkarah A, Ravoori MK, Shahzad MM, Lee JW, Mora E, Langley RR, Carroll AR, Matsuo K, Spannuth WA, Schmandt R, Jennings NB, Goodman BW, Jaffe RB, Nick AM, Kim HS, Guven EO, Chen YH, Li LY, Hsu MC, Coleman RL, Calin GA, Denkbas EB, Lim JY, Lee JS, Kundra V, **Birrer MJ,** Hung MC, Lopez-Berestein G, Sood AK. Regulation of tumor angiogenesis by EZH2 Cancer Cell. 2010 Aug 9;18(2):185-97.

200. Kikuchi J, Kinoshita I, Shimizu Y, Kikuchi E, Takeda K, Aburatani H, Oizumi S, Konishi J, Kaga K, Matsuno Y, **Birrer MJ**, Nishimura M, Dosaka-Akita H. Minichromosome maintenance (MCM) protein 4 as a marker for proliferation and its clinical and clinicopathological significance in non-small cell lung cancer. Lung Cancer. 2011 May;72(2):229-37.

201. Ghosh S, Albitar L, Lebaron R, Welch WR, Samimi G, **Birrer MJ**, Berkowitz RS, Mok SC. Up-

regulation of stromal versican expression in advanced state serous ovarian cancer. Gynecol Oncol. 2010 Oct;119(1):114-20.

202. Goode EL, Chenevix-Trench G, Song H, Ramus SJ, Notaridou M, Lawrenson K, Widschwendter M, Vierkant RA, Larson MC, Kjaer SK, **Birrer MJ**, Berchuck A, Schildkraut J, Tomlinson I, Kiemeney LA, Cook LS, Gronwald J, Garcia-Closas M, Gore ME, Campbell I, Whittemore AS, Sutphen R, Phelan C, Anton-Culver H, Pearce CL, Lambrechts D, Rossing MA, Chang-Claude J, Moysich KB, Goodman MT, Dörk T, Nevanlinna H, Ness RB, Rafnar T, Hogdall C, Hogdall E, Fridley BL, Cunningham JM, Sieh W, McGuire V, Godwin AK, Cramer DW, Hernandez D, Levine D, Lu K, Iversen ES, Palmieri RT, Houlston R, van Altena AM, Aben KK, Massuger LF, Brooks-Wilson A, Kelemen LE, Le ND, Jakubowska A, Lubinski J, Medrek K, Stafford A, Easton DF, Tyrer J, Bolton KL, Harrington P, Eccles D, Chen A, Molina AN, Davila BN, Arango H, Tsai YY, Chen Z, Risch HA, McLaughlin J, Narod SA, Ziogas A, Brewster W, Gentry-Maharaj A, Menon U, Wu AH, Stram DO, Pike MC; Wellcome Trust Case-Control Consortium, Beesley J, Webb PM; Australian Cancer Study (Ovarian Cancer); Australian Ovarian Cancer Study Group; Ovarian Cancer Association Consortium (OCAC). A genome- wide association study identifies susceptibility loci for ovarian cancer at 2q31 and 8q24. Nat Genet. 2010 Oct;42(10):874-9.

203. Bolton KL, Tyrer J, Song H, Ramus SJ, Notaridou M, Jones C, Sher T, Gentry-Maharaj A, Wozniak E, Tsai YY, Weidhaas J, Paik D, Van Den Berg DJ, Stram DO, Pearce CL, Wu AH, Brewster W, Anton-Culver H, Ziogas A, Narod SA, Levine DA, Kaye SB, Brown R, Paul J, Flanagan J, Sieh W, McGuire V, Whittemore AS, Campbell I, Gore ME, Lissowska J, Yang HP, Medrek K, Gronwald J, Lubinski J, Jakubowska A, Le ND, Cook LS, Kelemen LE, Brook-Wilson A, Massuger LF, Kiemeney LA, Aben KK, van Altena AM, Houlston R, Tomlinson I, Palmieri RT, Moorman PG, Schildkraut J, Iversen ES, Phelan C, Vierkant RA, Cunningham JM, Goode EL, Fridley BL, Kruger-Kjaer S, Blaeker J, Hogdall E, Hogdall C, Gross J, Karlan BY, Ness RB, Edwards RP, Odunsi K, Moyisch KB, Baker JA, Modugno F, Heikkinenen T, Butzow R, Nevanlinna H, Leminen A, Bogdanova N, Antonenkova N, Doerk T, Hillemanns P, Dürst M, Runnebaum I, Thompson PJ, Carney ME, Goodman MT, Lurie G, Wang-Gohrke S, Hein R, Chang-Claude J, Rossing MA, Cushing-Haugen KL, Doherty J, Chen C, Rafnar T, Besenbacher S, Sulem P, Stefansson K, **Birrer MJ**, Terry KL, Hernandez D, Cramer DW, Vergote I, Amant F, Lambrechts D, Despierre E, Fasching PA, Beckmann MW, Thiel FC, Ekici AB, Chen X; Australian Ovarian Cancer Study Group; Australian Cancer Study (Ovarian Cancer); Ovarian Cancer Association Consortium, Johnatty SE, Webb PM, Beesley J, Chanock S, Garcia-Closas M, Sellers T, Easton DF, Berchuck A, Chenevix-Trench G, Pharoah PD, Gayther SA. Common variants at 19p13 are associated with susceptibility to ovarian cancer. Nat Genet. 2010 Oct;42(10):880-4

204. **Birrer MJ**. The origin of ovarian cancer—is it getting clearer? N Engl J Med. 2010 Oct 14;363(16):1574-5.

205. Trimble EL, **Birrer MJ**, Hoskins WJ, Marth C, Petryshyn R, Quinn M, Thomas GM, Kitchener HC. Current academic clinical trials in ovarian cancer: Gynecologic Cancer Intergroup (GCIG) and United States National Cancer Institute (NCI) clinical trials planning meeting, May, 2009. Int J Gynecol Cancer. 2010 Oct;20(7):1290-8.

206. Matei D, Sill MW, Lankes HA, Degeest K, Bristow RE, Mutch D, Yamada SD, Cohn D, Calvert V,

Farley J, Petricoin EF, **Birrer MJ**. Activity of Sorafenib in Recurrent Ovarian Cancer and Primary Peritoneal Carcinomatosis: A Gynecologic Oncology Group Trial. Clin Oncol. 2011 Jan 1;29(1):69-75.

207. Emmanuel C, Gava N, Kennedy C, Balleine RL, Sharma R, Wain G, Brand A, Hogg R, Etemadmoghadam D, George J; Australian Ovarian Cancer Study Group, **Birrer MJ**, Clarke CL, Chenevix-Trench G, Bowtell DD, Harnett PR, deFazio A. Comparison of expression profiles in ovarian epithelium in vivo and ovarian cancer identifies novel candidate genes involved in disease pathogenesis. PLoS One. 2011 Mar 15;6(3):e17617.

208. Konstantinopoulos PA, Cannistra SA, Fountzilas H, Culhane A, Pillay K, Rueda B, Cramer D, Seiden M, **Birrer M**, Coukos G, Zhang L, Quackenbush J, Spentzos D. Integrated analysis of multiple microarray datasets identifies a reproducible survival predictor in ovarian cancer. PLoS One. 2011 Mar 29;6(3):e18202.

209. Helland A, Anglesio MS, George J, Cowin PA, Johnstone CN, House CM, Sheppard KE, Etemadmoghadam D, Melnyk N, Rustgi AK, Phillips WA, Johnsen H, Holm R, Kristensen GB, **Birrer MJ**, Australian Ovarian Cancer Study Group, Pearson RB, Børresen-Dale AL, Huntsman DG, Defazio A, Creighton CJ, Smyth GK, Bowtell DD. Deregulation of MYCN, LIN28B and LET7 in a Molecular Subtype of Aggressive High-Grade Serous Ovarian Cancers. PLoS One. 2011 Apr 13;6(4):e18064.

210. Anglesio MS, George J, Kulbe H, Friedlander M, Rischin D, Lemech C, Power J, Coward J, Cowin PA, House CM, Chakravarty P, Gorringe KL, Campbell IG; Australian Ovarian Cancer Study Group, Okamoto A, **Birrer MJ,** Huntsman DG, de Fazio A, Kalloger SE, Balkwill F, Gilks CB, Bowtell DD. IL6-STAT3-HIF signaling and therapeutic response to the angiogenesis inhibitor sunitinib in ovarian clear cell cancer. Clin Cancer Res. 2011 Apr 15;17(8):2538-48.

211. Ledermann JA, Marth C, Carey MS, **Birrer M**, Bowtell DD, Kaye S, McNeish I, Oza A, Scambia G, Rustin G, Stehman FB, Gershenson D, Thomas G, Berns E, Casado A, Ottevanger N, Hilpert F, Kim BG, Okamoto A, Bacon M, Kitchener H, Stuart GC; and on behalf of the Gynecologic Cancer InterGroup. Role of molecular agents and targeted therapy in clinical trials for women with ovarian cancer. Int J Gynecol Cancer. 2011 May 21(4):763-70.

212. Maritz MF, van der Watt PJ, Holderness N, **Birrer MJ**, Leaner VD. Inhibition of AP-1 suppresses cervical cancer cell proliferation and is associated with p21 expression. Biol Chem. 2011 May 392(5):439-48.

213. Kikuchi J, Kinoshita I, Shimizu Y, Kikuchi E, Takeda K, Aburatani H, Oizumi S, Konishi J, Kaga K, Matsuno Y, **Birrer MJ**, Nishimura M, Dosaka-Akita H. Minichromosome maintenance (MCM) protein 4 as a marker for proliferation and its clinical and clinicopathological significance in non-small cell lung cancer. Lung Cancer. 2011 May 72(2):229-37.

214. Farley J, Sill MW, **Birrer M**, Walker J, Schilder RJ, Thigpen JT, Coleman RL, Miller E, Rose PG, Lankes HA. Phase II study of cisplatin plus cetuximab in advanced, recurrent, and previously treated cancers of the cervix and evaluation of epidermal growth factor receptor

immunohistochemical expression: A Gynecologic Oncology Group study. Gynecol Oncol. 2011 May 1 121(2):303-8.

215. Farley J, Smith LM, Darcy KM, Brady MF, Bell J, McGuire W, **Birrer MJ**. Nuclear P27 expression in benign, borderline (LMP) and invasive tumors of the ovary and its association with prognosis: A gynecologic oncology group study. Gynecol Oncol. 2011 May 1;121(2):395-401.

216. King ER, Tung CS, Tsang YT, Zu Z, Lok GT, Deavers MT, Malpica A, Wolf JK, Lu KH, **Birrer MJ**, Mok SC, Gershenson DM, Wong KK. The Anterior Gradient Homolog 3 (AGR3) Gene Is Associated With Differentiation and Survival in Ovarian Cancer. Am J Surg Pathol. 2011 Jun;35(6):904-12.

217. Pharoah PD, Palmieri RT, Ramus SJ, Gayther SA, Andrulis IL, Anton-Culver H, Antonenkova N, Antoniou AC, Goldgar D; for the BCFR Investigators, Beattie MS, Beckmann MW, **Birrer MJ**, Bogdanova N, Bolton KL, Brewster W, Brooks-Wilson A, Brown R, Butzow R, Caldes T, Caligo MA, Campbell I, Chang-Claude J, Chen YA, Cook LS, Couch FJ, Cramer DW, Cunningham JM, Despierre E, Doherty JA, Dörk T, Dürst M, Eccles DM, Ekici AB, Easton D; for the EMBRACE Investigators, Fasching PA, de Fazio A, Fenstermacher DA, Flanagan JM, Fridley BL, Friedman E, Gao B, Sinilnikova O; for the GEMO Study Collaborators, Gentry-Maharaj A, Godwin AK, Goode EL, Goodman MT, Gross J, Hansen TV, Harnett P, Rookus M; for the HEBON Investigators, Heikkinen T, Hein R, Høgdall C, Høgdall E, Iversen ES, Jakubowska A, Johnatty SE, Karlan BY, Kauff ND, Kaye SB, Chenevix-Trench G; for the kConFab Investigators and the Consortium of Investigators of Modifiers of BRCA1/2, Kelemen LE, Kiemeney LA, Kjaer SK, Lambrechts D, Lapolla JP, Lázaro C, Le ND, Leminen A, Leunen K, Levine DA, Lu Y, Lundvall L, Macgregor S, Marees T, Massuger LF, McLaughlin JR, Menon U, Montagna M, Moysich KB, Narod SA, Nathanson KL, Nedergaard L, Ness RB, Nevanlinna H, Nickels S, Osorio A, Paul J, Pearce CL, Phelan CM, Pike MC, Radice P, Rossing MA, Schildkraut JM, Sellers TA, Singer CF, Song H, Stram DO, Sutphen R, Lindblom A; for the SWE-BRCA Investigators, Terry KL, Tsai YY, van Altena AM, Vergote I, Vierkant RA, Vitonis AF, Walsh C, Wang-Gohrke S, Wappenschmidt B, Wu AH, Ziogas A, Berchuck A, Risch HA; for the Ovarian Cancer Association Consortium. The Role of KRAS rs61764370 in Invasive Epithelial Ovarian Cancer: Implications for Clinical Testing. Clin Cancer Res. 2011 Jun 1;17(11):3742-3750.

218. Permuth-Wey J, Kim D, Tsai YY, Lin HY, Chen YA, Barnholtz-Sloan J, **Birrer MJ**, Bloom G, Chanock SJ, Chen Z, Cramer DW, Cunningham JM, Dagne G, Ebbert-Syfrett J, Fenstermacher D, Fridley BL, Garcia-Closas M, Gayther SA, Ge W, Gentry-Maharaj A, Gonzalez-Bosquet J, Goode EL, Iversen E, Jim H, Kong W, McLaughlin J, Menon U, Monteiro AN, Narod SA, Pharoah PD, Phelan CM, Qu X, Ramus SJ, Risch H, Schildkraut JM, Song H, Stockwell H, Sutphen R, Terry KL, Tyrer J, Vierkant RA, Wentzensen N, Lancaster JM, Cheng JQ, Sellers TA; Ovarian Cancer Association Consortium. LIN28B polymorphisms influence susceptibility to epithelial ovarian cancer. Cancer Res. 2011 Jun 1;71(11):3896-903.

219. Bodurka DC, Deavers MT, Tian C, Sun CC, Malpica A, Coleman RL, Lu KH, Sood AK, **Birrer MJ**, Ozols R, Baergen R, Emerson RE, Steinhoff M, Behmaram B, Rasty G, Gershenson DM. Reclassification of serous ovarian carcinoma by a 2-tier system: A Gynecologic Oncology Group Study. Cancer. 2012 Jun 15;118(12):3087-94.

220. Cancer Genome Atlas Research Network. Integrated genomic analyses of ovarian carcinoma. Nature. 2011 Jun 29;474(7353):609-15.

221. Stany MP, Vathipadiekal V, Ozbun L, Stone RL, Mok SC, Xue H, Kagami T, Wang Y, McAlpine JN, Bowtell D, Gout PW, Miller DM, Gilks CB, Huntsman DG, Ellard SL, Wang YZ, Vivas-Mejia P, Lopez-Berestein G, Sood AK, **Birrer MJ**. Identification of novel therapeutic targets in microdissected clear cell ovarian cancers. PLoS One. 2011;6(7):e21121

222. Righi E, Kashiwagi S, Yuan J, Santosuosso M, Leblanc P, Ingraham R, Forbes B, Edelblute B, Collette B, Xing D, Kowalski M, Mingari MC, Vianello F, **Birrer MJ**, Orsulic S, Dranoff G, Poznansky M. CXCL12/CXCR4 blockade induces multimodal anti-tumor effects and prolongs survival of immune competent mice with ovarian cancer. Cancer Res. 2011 Aug 15;71(16):5522-5534.

223. Hagemann AR, Cadungog M, Hagemann IS, Hammond R, Adams SF, Chu CS, Rubin S, Zhang L, Addya K, **Birrer MJ**, Gimotty PA, Coukos G. Tissue-based immune monitoring I: Tumor core needle biopsies allow in-depth interrogation of the tumor microenvironment. Cancer Biol Ther. 2011 Aug 15;12(4):357-66.

224. Hagemann AR, Hagemann IS, Cadungog M, Hwang WT, Patel P, Lal P, Hammond R, Gimotty PA, Chu CS, Rubin S, **Birrer MJ**, Powell DJ Jr, Feldman MD, Coukos G. Tissue-based immune monitoring II: Multiple tumor sites reveal immunologic homogeneity in serous ovarian carcinoma. Cancer Biol Ther. 2011 Aug 15;12(4):367-77.

225. Permuth-Wey J, Chen Z, Tsai YY, Lin HY, Chen YA, Barnholtz-Sloan J, **Birrer MJ**, Chanock SJ, Cramer DW, Cunningham JM, Fenstermacher D, Fridley BL, Garcia-Closas M, Gayther SA, Gentry-Maharaj A, Gonzalez Bosquet J, Iversen E, Jim H, McLaughlin J, Menon U, Narod SA, Phelan CM, Ramus SJ, Risch H, Song H, Sutphen R, Terry KL, Tyrer J, Vierkant RA, Wentzensen N, Lancaster JM, Cheng JQ, Berchuck A, Pharoah PD, Schildkraut JM, Goode EL, Sellers TA. MicroRNA processing and binding site polymorphisms are not replicated in the Ovarian Cancer Association Consortium. Cancer Epidemiol Biomarkers Prev. Cancer Epidemiol Biomarkers Prev. 2011 Aug;20(8):1793-1797.

226. Bitler BG, Nicodemus JP, Li H, Cai Q, Wu H, Hua X, Li T, **Birrer MJ**, Godwin AK, Cairns P, Zhang R. Wnt5a Suppresses Epithelial Ovarian Cancer by Promoting Cellular Senescence. Cancer Res. 2011 Oct 1;71(19):6184-94.

227. McCann CK, Growdon WB, Kulkarni-Datar K, Curley MD, Friel AM, Proctor JL, Sheikh H, Deyneko I, Ferguson JA, Vathipadiekal V, **Birrer MJ**, Borger DR, Mohapatra G, Zukerberg LR, Foster R, Macdougall JR, Rueda BR. Inhibition of hedgehog signaling antagonizes serous ovarian cancer growth in a primary xenograft model. PLoS One. 2011;6(11):e28077.

228. Burger RA, Brady MF, Bookman MA, Fleming GF, Monk BJ, Huang H, Mannel RS, Homesley HD, Fowler J, Greer BE, Boente M, **Birrer MJ**, Liang SX; Gynecologic Oncology Group. Incorporation of bevacizumab in the primary treatment of ovarian cancer. N Engl J Med. 2011 Dec

29;365(26):2473-83.

229. Vathipadiekal V, Saxena D, Mok SC, Hauschka PV, Ozbun L, **Birrer MJ**. Identification of a potential ovarian cancer stem cell gene expression profile from advanced stage papillary serous ovarian cancer. PLoS One. 2012;7(1):e29079.

230. White KL, Schildkraut JM, Palmieri RT, Iversen ES, Berchuck A, Vierkant RA, Rider DN, Charbonneau B, Cicek MS, Sutphen R, **Birrer MJ**, Pharoah PD, Song H, Tyrer J, Gayther SA, Ramus SJ, Wentzensen N, Yang HP, Garcia-Closas M, Phelan CM, Cunningham JM, Fridley BL, Sellers TA, Goode EL. Ovarian Cancer Risk Associated with Inherited Inflammation-Related Variants. Cancer Res. 2012 Mar 1;72(5):1064-1069.

231. Li H, Bitler BG, Vathipadiekal V, Maradeo ME, Slifker M, Creasy CL, Tummino PJ, Cairns P, **Birrer, MJ**, Zhang R. ALDH1A1 Is a Novel EZH2 Target Gene in Epithelial Ovarian Cancer Identified by Genome-Wide Approaches. Cancer Prev Res (Phila). 2012 Mar;5(3):484-91.

232. Stevens KN, Kelemen LE, Wang X, Fridley BL, Vierkant RA, Fredericksen Z, Armasu SM, Tsai YY, Berchuck A, Association Consortium OC, Narod SA, Phelan CM, Sutphen R, **Birrer MJ**, Schildkraut JM, Sellers TA, Goode EL, Couch FJ. Common variation in Nemo-like kinase (NLK) is associated with risk of ovarian cancer. Cancer Epidemiol Biomarkers Prev. 2012 Mar;21(3):523-528.

233. Del Carmen MG, **Birrer M**, Schorge JO. Carcinosarcoma of the ovary: A review of the literature. Gynecol Oncol. Gynecol Oncol. 2012 Apr;125(1):271-7.

234. Ferriss JS, Kim Y, Duska L, **Birrer M**, Levine DA, Moskaluk C, Theodorescu D, Lee JK. Multi-gene expression predictors of single drug responses to adjuvant chemotherapy in ovarian carcinoma: predicting platinum resistance. PLoS One. 2012;7(2):e30550.

235. Engler DA, Gupta S, Growdon WB, Drapkin RI, Nitta M, Sergent PA, Allred SF, Gross J, Deavers MT, Kuo WL, Karlan BY, Rueda BR, Orsulic S, Gershenson DM, **Birrer MJ**, Gray JW, Mohapatra G. Genome wide DNA copy number analysis of serous type ovarian carcinomas identifies genetic markers predictive of clinical outcome. PLoS One. 2012;7(2):e30996.

236. Kassler S, Donninger H, **Birrer MJ**, Clark GJ. RASSF1A and the Taxol Response in Ovarian Cancer. Mol Bio Int. 2012;263267.

237. Rubatt JM, Darcy KM, Tian C, Muggia F, Dhir R, Armstrong DK, Bookman MA, Niedernhofer LJ, Deloia J, **Birrer M**, Krivak TC. Pre-treatment tumor expression of ERCC1 in women with advanced stage epithelial ovarian cancer is not predictive of clinical outcomes: a Gynecologic Oncology Group study. Gynecol Oncol. 2012 May;125(2):421-6.

238. Alsop K, Fereday S, Meldrum C, Defazio A, Emmanuel C, George J, Dobrovic A, **Birrer MJ**, Webb PM, Stewart C, Friedlander M, Fox S, Bowtell D, Mitchell G. BRCA Mutation Frequency and Patterns of Treatment Response in BRCA Mutation-Positive Women With Ovarian Cancer: A Report From the Australian Ovarian Cancer Study Group. J Clin Oncol. 2012 Jul 20;30(21):2654-

63.

239. Del Carmen MG, **Birrer M**, Schorge JO. Clear cell carcinoma of the ovary: A review of the literature. Gynecol Oncol. 2012 Sep;126(3):481-90.

240. Del Carmen MG, **Birrer M**, Schorge JO. Uterine papillary serous cancer: A review of the literature. Gynecol Oncol. 2012 Dec; 127(3):651-61.

241. Sethi G, Pathak HB, Zhang H, Zhou Y, Einarson MB, Vathipadiekal V, Gunewardena S, **Birrer MJ**, Godwin AK. An RNA interference lethality screen of the human druggable genome to identify molecular vulnerabilities in epithelial ovarian cancer. PLoS One. 2012;7(10):e47086. doi: 10.1371/journal.pone.0047086. Epub 2012 Oct 9.

242. Li H, Cai Q, Wu H, Vathipadiekal V, Dobbin ZC, Li T, Hua X, Landen CN Jr, **Birrer MJ**, Sanchez-Beato M, Zhang R. SUZ12 promotes human epithelial ovarian cancer by suppressing apoptosis via silencing HRK. Mol Cancer Res. 2012 Nov; 10 (11): 1462-72

243. May T, Shoni M, Crum CP, Xian W, Vathipadiekal V, **Birrer M**, Rosen B, Tone A, Murphy KJ. Low-grade and high-grade serous Mullerian carcinoma: Review and analysis of publicly available gene expression profiles. Gynecol Oncol. 2012 Dec 16. S0090-8258(12)00956-0. 10.1016/j.ygyno.2012.12.009.

244. Verhaak RG, Tamayo P, Yang JY, Hubbard D, Zhang H, Creighton CJ, Fereday S, Lawrence M, Carter SL, Mermel CH, Kostic AD, Etemadmoghadam D, Saksena G, Cibulskis K, Duraisamy S, Levanon K, Sougnez C, Tsherniak A, Gomez S, Onofrio R, Gabriel S, Chin L, Zhang N, Spellman PT, Zhang Y, Akbani R, Hoadley KA, Kahn A, Köbel M, Huntsman D, Soslow RA, Defazio A, **Birrer MJ**, Gray JW, Weinstein JN, Bowtell DD, Drapkin R, Mesirov JP, Getz G, Levine DA, Meyerson M.Prognostically relevant gene signatures of high-grade serous ovarian carcinoma.J Clin Invest. 2013 Jan 2;123(1):517-25.

245. Zaid T, Yeung TL, Thompson MS, Leung C, Harding T, Co N, Schmandt RE, Kwan SY, Rodriguez-Aguayo C, Lopez-Berestein G, Sood AK, Wong KK, **Birrer MJ**, Mok SC Identification of FGFR4 as a Potential Therapeutic Target for Advanced-Stage High-grade Serous Ovarian Cancer. Clin Cancer Res. 2013 Jan 29.

246. Farley J, Brady WE, Vathipadiekal V, Lankes HA, Coleman R, Morgan MA, Mannel R, Yamada SD, Mutch D, Rodgers WH, **Birrer M**, Gershenson DM Selumetinib in women with recurrent low-grade serous carcinoma of the ovary or peritoneum: an open-label, single-arm, phase 2 study. Lancet Oncol. 2013 Feb;14(2):134-140.

247. Permuth-Wey J, Lawrenson K, Shen HC, Velkova A, Tyrer JP, Chen Z, Lin HY, Ann Chen Y, Tsai YY, Qu X, Ramus SJ, Karevan R, Lee J, Lee N, Larson MC, Aben KK, Anton-Culver H, Antonenkova N, Antoniou AC, Armasu SM; stralian Cancer Study; stralian Ovarian Cancer Study, Bacot F, Baglietto L, Bandera EV, Barnholtz-Sloan J, Beckmann MW, **Birrer MJ**, Bloom G, Bogdanova N, Brinton LA, Brooks-Wilson A, Brown R, Butzow R, Cai Q, Campbell I, Chang-Claude J Chanock S, Chenevix-Trench G, Cheng JQ, Cicek MS, Coetzee GA; Consortium of

Investigators of Modifiers of BRCA1/2, Cook LS, Couch FJ, Cramer DW, Cunningham JM, Dansonka-Mieszkowska A, Despierre E, Doherty JA, Dörk T, du Bois A, Dürst M, Easton DF, Eccles D, Edwards R, Ekici AB, Fasching PA, Fenstermacher DA, Flanagan JM, Garcia-Closas M, Gentry-Maharaj A, Giles GG, Glasspool RM, Gonzalez-Bosquet J, Goodman MT, Gore M, Górski B, Gronwald, J, Hall P, Halle MK, Harter P, Heitz F, Hillemanns P, Hoatlin M, Høgdall CK, Høgdall E, Hosono S, Jakubowska A, Jensen A, Jim H, Kalli KR, Karlan BY, aye SB, Kelemen LE, Kiemeney LA, Kikkawa F, Konecny GE, Krakstad C, Krüger Kjaer S, Kupryjanczyk J, Lambrechts D, Lambrechts S, Lancaster JM, Le ND, Leminen A, Levine DA, Liang D, Kiong Lim B, Lin J, Lissowska J, Lu KH, Lubiński J, Lurie G, Massuger LF, Matsuo K, McGuire V, McLaughlin JR, Menon U, Modugno F, Moysich KB, Nakanishi T, Narod SA, Nedergaard L, Ness RB, Nevanlinna H, Nickels S, Noushmehr H, Odunsi K, Olson SH, Orlow I, Paul J, Pearce CL, Pejovic T, Pelttari LM, Pike MC, Poole EM, Raska P, Renner SP, Risch HA, Rodriguez-Rodriguez L, Anne Rossing M, Rudolph A, Runnebaum IB, Rzepecka IK, Salvesen HB, Schwaab I, Severi G, Shridhar V, Shu XO, Shvetsov YB, Sieh W, Song H, Southey MC, Spiewankiewicz B, Stram D, Sutphen R, Teo SH, Terry KL, Tessier DC, Thompson PJ, Tworoger SS, van Altena AM, Vergote I, Vierkant RA, Vincent D, Vitonis AF, Wang-Gohrke S, Palmieri Weber R, Wentzensen N, Whittemore AS, Wik E, Wilkens LR, Winterhoff B, Ling Woo Y, Wu AH, Xiang YB, Yang HP, Zheng W, Ziogas A, Zulkifli F, Phelan CM, Iversen E, Schildkraut JM, Berchuck A, Fridley BL, Goode EL, Pharoah PD, Monteiro AN, Sellers TA, Gayther SA. Identification and molecular characterization of a new ovarian cancer susceptibility locus at 17q21.31. Nat Commun. 2013 Mar 27;4:1627

248. Ganzfried BF, Riester M, Haibe-Kains B, Risch T, Tyekucheva S, Jazic I, Wang XV, Ahmadifar M, **Birrer MJ**, Parmigiani G, Huttenhower C, Waldron L. CuratedOvarianData: Clinically annotated data for the ovarian cancer transcriptome. Database (Oxford). 2013 Apr 2;2013

249. Pharoah PD, Tsai YY, Ramus SJ, Phelan CM, Goode EL, Lawrenson K, Buckley M, Fridley BL, Tyrer JP, Shen H, Weber R, Karevan R, Larson MC, Song H, Tessier DC, Bacot F, Vincent D, Cunningham JM, Dennis J, Dicks E; stralian Cancer Study; stralian Ovarian Cancer Study Group, Aben KK, Anton-Culver H, Antonenkova N, Armasu SM, Baglietto L, Bandera EV, Beckmann MW, **Birrer MJ**, Bloom G, Bogdanova N, Brenton JD, Brinton LA, Brooks-Wilson A, BrownR, Butzow R, Campbell I, Carney ME, Carvalho RS, Chang-Claude J, Chen YA, Chen Z, Chow WH, Cicek MS, Coetzee G, Cook LS, Cramer DW, Cybulski C, Dansonka-Mieszkowska A, Despierre E, Doherty JA, Dörk T, du Bois A, Dürst M, Eccles D, Edwards R, Ekici AB, Fasching PA, Fenstermacher D, Flanagan J, Gao YT, Garcia-Closas M, Gentry-Maharaj A, Giles G, Gjyshi A, Gore M, Gronwald J, Guo Q, Halle MK, Harter P, Hein A, Heitz F, Hillemanns P, Hoatlin M, Høgdall E, Høgdall CK, Hosono S, Jakubowska A, Jensen A, Kalli KR, Karlan BY, Kelemen LE, Kiemeney LA, Kjaer SK, Konecny GE, Krakstad C, Kupryjanczyk J, Lambrechts D, Lambrechts S, Le ND, Lee N, Lee J, Leminen A, Lim BK, Lissowska J, Lubiński J, Lundvall L, Lurie G, Massuger LF, Matsuo K, McGuire V, McLaughlin JR, Menon U, Modugno F, Moysich KB, Nakanishi T, Narod SA, Ness RB, Nevanlinna H, Nickels S, Noushmehr H, Odunsi K, Olson S, Orlow I, Paul J, Pejovic T, Pelttari LM, Permuth-Wey J, Pike MC, Poole EM, Qu X, Risch HA, Rodriguez-Rodriguez L, Rossing MA, Rudolph A, Runnebaum I, Rzepecka IK, Salvesen HB, Schwaab I, Severi G, Shen H, Shridhar V, Shu XO, Sieh W, Southey MC, Spellman P, Tajima K, Teo SH, Terry KL, Thompson PJ, Timorek A, Tworoger SS, van Altena AM, van den Berg D, Vergote I, Vierkant RA, Vitonis AF, Wang-Gohrke S, Wentzensen N, Whittemore AS, Wik E, Winterhoff B, Woo YL, Wu AH, Yang HP, Zheng W, Ziogas A, Zulkifli F, Goodman MT, Hall P, Easton DF,

Pearce CL, Berchuck A, Chenevix-Trench G, Iversen E, Monteiro AN, Gayther SA, Schildkraut JM, Sellers TA. GWAS meta-analysis and replication identifies three new susceptibility loci for ovarian cancer. Nat Genet. 2013 Apr;45(4):362-70.

250. Lesnock JL, Darcy KM, Tian C, Deloia JA, Thrall MM, Zahn C, Armstrong DK, **Birrer MJ**, Krivak TC. BRCA1 expression and improved survival in ovarian cancer patients treated with intraperitoneal cisplatin and paclitaxel: a Gynecologic Oncology Group Study. Br J Cancer. 2013 Apr 2;108(6):1231-7.

251. Goode EL, DeRycke M, Kalli KR, Oberg AL, Cunningham JM, Maurer MJ, Fridley BL, Armasu SM, Serie DJ, Ramar P, Goergen K, Vierkant RA, Rider DN, Sicotte H, Wang C, Winterhoff B, Phelan CM, Schildkraut JM, Weber RP, Iversen E, Berchuck A, Sutphen R, **Birrer MJ**, Hampras S, Preus L, Gayther SA, Ramus SJ, Wentzensen N, Yang HP, Garcia-Closas M, Song H, Tyrer J, Pharoah PP, Konecny G, Sellers TA, Ness RB, Sucheston LE, Odunsi K, Hartmann LC, Moysich KB, Knutson KL. Inherited variants in regulatory T cell genes and outcome of ovarian cancer. PLoS One. 2013;8(1):e53903.

252. Cancer Genome Atlas Research Network, Kandoth C, Schultz N, Cherniack AD, Akbani R, Liu Y, Shen H, Robertson AG, Pashtan I, Shen R, Benz CC, Yau C, Laird PW, Ding L, Zhang W, Mills GB, Kucherlapati R, Mardis ER, Levine DA. Integrated genomic characterization of endometrial carcinoma. Nature. 2013 May 2;497(7447):67-73.

253. Bradford LS, Rauh-Hain JA, Schorge J, **Birrer MJ**, Dizon DS. Advances in the Management of Recurrent Endometrial Cancer. Am J Clin Oncol. 2013 Jun 11; PubMed PMID:23764681

254. Holderness Parker N, Donninger H, **Birrer MJ**, Leaner VD. p21-activated kinase 3 (PAK3) is an AP-1 regulated gene contributing to actin organisation and migration of transformed fibroblasts. PLoS One. 2013 Jun 20;8(6):e66892. doi:10.1371/journal.pone.0066892. Print 2013. PubMed PMID: 23818969

255. Yeung TL, Leung CS, Wong KK, Samimi G, Thompson MS, Liu J, Zaid TM, Ghosh S, **Birrer MJ**, Mok SC. TGF-b modulates ovarian cancer invasion by upregulating CAF-derived versican in the tumor micro-environment. Cancer Res. 2013 Aug 15;73(16):5016-28. doi: 10.1158/0008-5472.CAN-13-0023. Epub 2013 Jul 3. PubMed PMID: 23824740; PubMed Central PMCID: PMC3745588.

256. Liu JF, Tolaney SM, **Birrer M**, Fleming GF, Buss MK, Dahlberg SE, Lee H, Whalen C, Tyburski K, Winer E, Ivy P, Matulonis UA. A Phase 1 trial of the poly(ADP-ribose) polymerase inhibitor olaparib (AZD2281) in combination with the anti-angiogenic cediranib (AZD2171) in recurrent epithelial ovarian or triple-negative breast cancer. Eur J Cancer. 2013 Sep;49(14):2972-8. doi:10.1016/j.ejca.2013.05.020. Epub 2013 Jun 27. PubMed PMID: 23810467.

257. He L, Guo L, Vathipadiekal V, Sergent PA, Growdon WB, Engler DA, Rueda BR, **Birrer MJ**, Orsulic S, Mohapatra G. Identification of LMX1B as a novel oncogene in human ovarian cancer. Oncogene. 2013 Sep 23. doi: 10.1038/onc.2013.375. PubMed PMID: 24056967.

258. Rauh-Hain JA, Guseh SH, Esselen KM, Growdon WB, Schorge JO, Horowitz NS, Krasner CN, del Carmen MG, **Birrer MJ**, Dizon DS. Patterns of recurrence in patients treated with bevacizumab in the primary treatment of advanced epithelial ovarian cancer. Int J Gynecol Cancer. 2013 Sep;23(7):1219-25.

259. Penson RT, Dizon DS, **Birrer MJ**. Clear cell cancer of the ovary. Curr Opin Oncol. 2013 Sep;25(5):553-7. doi: 10.1097/CCO.0b013e328363e0c7. PubMed PMID: 23942300.

260. Levanon K, Sapoznik S, Bahar-Shany K, Brand H, Shapira-Frommer R, Korach J, Hirsch MS, Roh MH, Miron A, Liu JF, Vena N, Ligon AH, Fotheringham S, Bailey D, Flavin RJ, **Birrer MJ**, Drapkin RI. FOXO3a loss is a frequent early event in high-grade pelvic serous carcinogenesis. Oncogene. 2013 Sep 30. doi:10.1038/onc.2013.394. PubMed PMID: 24077281.

261. Wei W, Mok SC, Oliva E, Kim SH, Mohapatra G, **Birrer MJ**. FGF18 as a prognostic and therapeutic biomarker in ovarian cancer. J Clin Invest. 2013 Oct 1;123(10):4435-48. doi: 10.1172/JCI70625. Epub 2013 Sep 9. PubMed PMID: 24018557; PubMed Central PMCID: PMC3784549.

262. Creutzberg CL, Kitchener HC, **Birrer MJ**, Landoni F, Lu KH, Powell M, Aghajanian C, Edmondson R, Goodfellow PJ, Quinn M, Salvesen HB, Thomas G. GCIG EndometrialCancer Clinical Trials Planning Meeting. Gynecologic Cancer InterGroup (GCIG) endometrial cancer clinical trials planning meeting: taking endometrial cancer trials into the translational era. Int J Gynecol Cancer. 2013 Oct;23(8):1528-34. doi: 10.1097/IGC.0b013e3182a26edb. PubMed PMID: 24257568.

263. Dizon DS, Ambrosio AJ, Feltmate C, Wright AA, Campos SM, Viswanathan A. Cervical Cancer Special Interest Group of Dana-Farber/Harvard Comprehensive Cancer Center Gynecologic Cancer Management Team. Pushing the bar in treatment of cervical cancer: what can comprehensive cancer centers do on their own? Gynecol Oncol. 2013 Nov;131(2):464-6. doi: 10.1016/j.ygyno.2013.08.002. Epub 2013 Aug 9. PubMed PMID: 23938373.

264. Tanwar PS, Mohapatra G, Chiang S, Engler DA, Zhang L, Kaneko-Tarui T, Ohguchi Y, **Birrer MJ**, Teixeira JM. Loss of LKB1 and PTEN tumor suppressor genes in the ovarian surface epithelium induces papillary serous ovarian cancer. Carcinogenesis. 2013 Dec 2. PubMed PMID: 24170201.

265. Peterson VM, Castro CM, Chung J, Miller NC, Ullal AV, Castano MD, Penson RT, Lee H, **Birrer MJ**, Weissleder R. Ascites analysis by a microfluidic chip allows tumor-cell profiling. Proc Natl Acad Sci U S A. 2013 Dec 17;110(51):E4978-86. doi:10.1073/pnas.1315370110. Epub 2013 Dec 2. PubMed PMID: 24297935.

266. Wei W, Dizon D, Vathipadiekal V, **Birrer MJ**. Ovarian cancer: genomic analysis. Ann Oncol. 2013 Dec; 24 Suppl 10:x7-x15. doi: 10.1093/annonc/mdt462. PubMed PMID: 24265410; PubMed Central PMCID: PMC3836569.

267. Jeong W, Kim SB, Sohn BH, Park YY, Park ES, Kim SC, Kim SS, Johnson RL, **Birrer M**, Bowtell DS, Mills GB, Sood A, Lee JS. Activation of YAP1 Is Associated with Poor Prognosis and

Response to Taxanes in Ovarian Cancer. Anticancer Res. 2014 Feb;34(2):811-7. PubMed PMID: 24511017

268. Bookman MA, Gilks CB, Kohn EC, Kaplan KO, Huntsman D, Aghajanian C, **Birrer MJ**, Ledermann, JA, Oza AM, Sweneron KD. Better therapeutic trials in ovarian cancer. J Natl Cancer Inst. 2014 April 1;106(4).

269. Riester M, Wei W, Waldron L, Culhane AC, Trippa L, Oliva E, Kim S, Michor F, Hutterenhower C, Parmigiani G, **Birrer MJ**. Risk prediction for late-stage ovarian cancer by meta-analysis of 1525 patient samples. J Natl Cancer Instit. 2014 April 3;106(5): dju 048. April 3 Pubmed PMID: 24700803.

270. Waldron L, Haibe-Kains B, Culhane AC, Riester M, Ding J, Wang XV, Ahmadifar M, Tyekucheva S, Bernau C, Risch T, Ganzfried BF, Hutterhower C, **Birrer MJ**, Parmigiani G. Comparative meta-analysis of prognostic gene signatures for late-stage ovarian cancer. J Natl Cancer Instit. 2014 April 3;106(5): dju 049.

271. **Birrer MJ**. Ovarian cancer: targeting the untargetable. Am Soc Clin Oncol Educ Book. 2014;34:13-5.

272. Dizon DS, **Birrer MJ**. Advances in the diagnosis and treatment of uterine sarcomas. Disc Med. 2014 Jun;17(96):339-45.

273. Leung CS, Yeung TL, Yip KP, Pradeep S, Balasubramanian L, Liu J, Wong KK, Mangala LS, Armaiz-Pena GN, Lopez-Berextein G, Sood AK, **Birrer MJ**, Mok SC. Calcium dependent FAK/CREB/TNNC1 signaling mediates the effect of stromal MFAP% on ovarian cancer metastatic potential. Nat Commun 2014, Oct 3;5:5092. PMCID: PMC4185407.

274. Matulonis U, Vergote I, Backes F, Martin LP, McMeekin S, **Birrer M**, Campana F, Xu Y, Egile C, Ghamande S. Phase II study of the P13K inhibitor pilaralisib (SAR245408; XL147) in patients with advanced or recurrent endometrial carcinoma. Gynecol Oncol. 2014 Dec 17. pii: S0090-8258(14)01574-1. doi: 10.1016/j.ygyno.2014.12.019.

275. Ghosh D, Bagley AF, NaYJ, **Birrer MJ**, Bhatia SN, Beleher AM. Deep, noninvasive imagine and surgical guidance of submillimeter tumors using targeted M13-stabilized single-walled carbon nanotubes. Proc Natl Acad Sci USA. 2014 Sep 23;111(38):13948-53. doi:10.1073/pnas.1400821111. Epub 2014 Sept. 1.

276. Liu JF, Barry WT, **Birrer M**, Lee JM, Buckanovich RJ, Fleming GF, Rimel B, Buss MK, Nattam S, Hurteau J, Luo W, Quy P, Whalen C, Obermayer L, Lee H, Winer EP, Kohn EC, Ivy SP, Matulonis UA. Lancet Oncol. 2014 Oct. 15(11):1207-14. doi: 10.1016/S1470-2045(14)70391-2. Epub 2014 Sept 10.

277. Waldron L, Reister M, **Birrer MJ**. Molecular subtypes of high-grade serous ovarian cancer: the holy grail? J Natl Cancer Inst. 2014 Sept. 30;106(10). Pii: dju297. doi: 210.1093/jcni/dju297. Print 2014 Oct.

278. Pham E, **Birrer MJ**, Eliasof S, Garmey E, Lazarus D, Lee CR, Man S, Matulonis UA, Peters CG, Xu P, Krasner C, Kerbel RS. Translational impact of nanoparticle-drug conjugate CRLX101 with or without bevacizumab in advanced ovarian cancer. Clin Cancer Res. 2015 Feb 15; 21(4):808-18.

279. Matulonis U, Vergote I, Backes F, Martin LP, McMeekin S, **Birrer M**, Campana F, Xu Y, Egile C, Ghamande S. Phase II study of the PI3K inhibitor pilaralisib (SAR245408; XL147) in patients with advanced or recurrent endometrial carcinoma. Gynecol Oncol. 2015 Feb;136(2):246-53. doi: 10.1016/j.ygyno.2014.12.019. Epub 2014 Dec 17.

280. Castro CM, Im H, Le C, Lee H, Weissleder R, **Birrer MJ**. Exploring alternative ovarian cancer biomarkers using innovative nanotechnology strategies. Cancer Metastasis Rev. 2015 Mar;34(1):75-82. doi: 10.1007/s10555-014-9546-9.

281. Tyekucheva S, Martin NE, Stack EC, Wei W, Vathipadiekal V, Waldron L, Fiorentino M, Lis RT, Stampfer MJ, Loda M, Parmigiani G, Mucci LA, **Birrer M**. Comparing Platforms for Messenger RNA Expression Profiling of Archival Formalin-Fixed, Paraffin-Embedded Tissues. J Mol Diagn. 2015 Jul;17(4):374-81. doi: 10.1016/j.jmoldx.2015.02.002. Epub 2015 Apr 30.

282. Vathipadiekal V, Wang V, Wei W, Waldron L, Drapkin R, Gillette M, Skates S, **Birrer M**. Creation of a Human Secretome: A Novel Composite Library of Human Secreted Proteins: Validation Using Ovarian Cancer Gene Expression Data and a Virtual Secretome Array. Clin Cancer Res. 2015 May 5

283. Elagib KE, Xiao M, Hussaini IM, Delehanty LL, Palmer LA, Racke FK, **Birrer MJ**, Ganapathy-Kanniappan S, McDevitt MA, Goldfarb AN. Erratum for Elagib et al., Jun blockade of erythropoiesis: role for repression of GATA-1 by HERP2. Mol Cell Biol. 2015 Jun;35(12):2215. doi: 10.1128/MCB.00374-15.

284. Wei W, **Birrer MJ**. Spleen Tyrosine Kinase Confers Paclitaxel Resistance in Ovarian Cancer. Cancer Cell. 2015 Jul 13;28(1):7-9. doi: 10.1016/j.ccell.2015.06.008.

285. Pépin D, Sosulski A, Zhang L, Wang D, Vathipadiekal V, Hendren K, Coletti CM, Yu A, Castro, CM, **Birrer MJ**, Gao G, Donahoe PK. AAV9 delivering a modified human Mullerian inhibiting substance as a gene therapy in patient-derived xenografts of ovarian cancer. Proc Natl Acad Sci U S A. 2015 Aug 11;112(32):E4418-27. doi: 10.1073/pnas.1510604112. Epub 2015 Jul 27.

286. Richards EJ, Permuth-Wey J, Li Y, Chen YA, Coppola D, Reid BM, Lin HY, Teer JK, Berchuck A, **Birrer MJ**, Lawrenson K, Monteiro AN, Schildkraut JM, Goode EL, Gayther SA, Sellers TA, Cheng JQ. A functional variant in HOXA11-AS, a novel long non-coding RNA, inhibits the oncogenic phenotype of epithelial ovarian cancer. Oncotarget. 2015 Sep 22.

287. Bowtell DD, Böhm S, Ahmed AA, Aspuria PJ, Bast RC Jr, Beral V, Berek JS, **Birrer MJ**, Blagden S, Bookman MA, Brenton JD, Chiappinelli KB, Martins FC, Coukos G, Drapkin R, Edmondson R, Fotopoulou C, Gabra H, Galon J, Gourley C, Heong V, Huntsman DG, Iwanicki M, Karlan BY, Kaye A, Lengyel E, Levine DA, Lu KH, McNeish IA, Menon U, Narod SA, Nelson BH, Nephew

KP, Pharoah P, Powell DJ Jr, Ramos P, Romero IL, Scott CL, Sood AK, Stronach EA, Balkwill FR. Rethinking ovarian cancer II: reducing mortality from high-grade serous ovarian cancer. Nat Rev Cancer. 2015 Oct 23;15(11):668-79. doi: 10.1038/nrc4019.

288. Sarantopoulos J, Mita MM, **Birrer MJ**, Cranmer LD, Campos LT, Zhang X, Bristow P, Kaito H, Strout V, Camacho LH. Phase 1 Study of monotherapy with KHK2866, an anti-heparin-binding erpidermal growth factor-like growth factor monoclonal antibody, in patients with advanced cancer. Target Oncol. 2015 Oct 27.

289. Ye H, Tanenbaum LM, Na YJ, Mantzavinou A, Fulci G, Del Carmen MG, **Birrer MJ**, Cima MJ. Sustained, low-dose intraperitoneal cisplatin improves treatment outcome in ovarian cancer mouse models. J Control Release. 2015 Dec 28;220(Pt A):358-67. doi: 10.1016/j.jconrel.2015.11.001. Epub 2015 Nov 5.

290. Moran-Jones K, Gloss BS, Murali R, Chang DK, Colvin EK, Jones MD, Yuen S, Howell VM, Brown LM, Wong CW, Spong SM, Scarlett CJ, Hacker NF, Ghosh S, Mok SC, **Birrer MJ**, Samimi G. Connective tissue growth factor as a novel therapeutic target in high grade serous ovarian cancer. Oncotarget. 2015 Nov 11. doi: 10.18632/oncotarget.6082.

291. Bolton KL, Tyrer J, Song H, Ramus SJ, Notaridou M, Jones C, Sher T, Gentry-Maharaj A, Wozniak E, Tsai YY, Weidhaas J, Paik D, Van Den Berg DJ, Stram DO, Pearce CL, Wu AH, Brewster W, Anton-Culver H, Ziogas A, Narod SA, Levine DA, Kaye SB, Brown R, Paul J, Flanagan J, Sieh W, McGuire V, Whittemore AS, Campbell I, Gore ME, Lissowska J, Yang HP, Medrek K, Gronwald J, Lubinski J, Jakubowska A, Le ND, Cook LS, Kelemen LE, Brooks-Wilson A, Massuger LF, Kiemeney LA, Aben KK, van Altena AM, Houlston R, Tomlinson I, Palmieri RT, Moorman PG, Schildkraut J, Iversen ES, Phelan C, Vierkant RA, Cunningham JM, Goode EL, Fridley BL, Kruger-Kjaer S, Blaeker J, Hogdall E, Hogdall C, Gross J, Karlan BY, Ness RB, Edwards RP, Odunsi K, Moyisch KB, Baker JA, Modugno F, Heikkinenen T, Butzow R, Nevanlinna H, Leminen A, Bogdanova N, Antonenkova N, Doerk T, Hillemanns P, Dürst M, Runnebaum I, Thompson PJ, Carney ME, Goodman MT, Lurie G, Wang-Gohrke S, Hein R, Chang-Claude J, Rossing MA, Cushing-Haugen KL, Doherty J, Chen C, Rafnar T, Besenbacher S, Sulem P, Stefansson K, **Birrer MJ**, Terry KL, Hernandez D, Cramer DW, Vergote I, Amant F, Lambrechts D, Despierre E, Fasching PA, Beckmann MW, Thiel FC, Ekici AB, Chen X; Australian Ovarian Cancer Study Group, Johnatty SE, Webb PM, Beesley J, Chanock S, Garcia-Closas M, Sellers T, Easton DF, Berchuck A, Chenevix-Trench G, Pharoah PD, Gayther SA. Corrigendum: Common variants at 19p13 are associated with susceptibility to ovarian cancer. Nat Genet. 2015 Dec 29;48(1):101. doi: 10.1038/ng0116-101b.

292. Norquist BM, Harrell MI, Brady MF, Walsh T, Lee MK, Gulsuner S, Bernards SS, Casadei S, Yi Q, Burger RA, Chan JK, Davidson SA, Mannel RS, DiSilvestro PA, Lankes HA, Ramirez NC, King MC, Swisher EM, **Birrer MJ**. Inherited mutations in women with ovarian carcinoma.JAMA Oncol. 2015 Dec 30:1-9. doi: 10.1001/jamaoncol.2015.5495.

293. van der Watt PJ, Chi A, Stelma T, Stowell C, Strydom E, Carden S, Angus L, Hadley K, Lang D, Wei W, **Birrer MJ**, Trent JO, Leaner VD. Targeting the nuclear import receptor, Kpnβ1 as an anti-cancer therapeutic. Mol Cancer Ther. 2016 Feb 1. pii: molcanther.0052.2015.

294. Dizon DS, **Birrer MJ**. Making a difference: Distinguishing two primaries from metastasis in synchronous tumors of the ovary and uterus. J Natl Cancer Inst. 2016 Feb 1;108(6). pii: djv442. doi: 10.1093/jnci/djv442. Print 2016 Jun.

295. Chan JK, Brady MF, Penson RT, Huang H, **Birrer MJ**, Walker JL, DiSilvestro PA, Rubin SC, Martin LP, Davidson SA, Huh WK, O'Malley DM, Boente MP, Michael H, Monk BJ. Weekly vs. every-3-week paclitaxel and carboplatin for ovarian cancer. N Engl J Med. 2016 Feb 25;374(8):738-48. doi: 10.1056/NEJMoa1505067.

296. Swisher EM, Harrell MI, Norquist BM, Walsh T, Brady M, Lee M, Hershberg R, Kalli KR, Lankes H, Konnick EQ, Pritchard CC, Monk BJ, Chan JK, Burger R, Kaufmann SH, **Birrer MJ**. Somatic mosaic mutations in PPM1D and TP53 in the blood of women with ovarian carcinoma. JAMA Oncol. 2016 Feb 4. doi: 10.1001/jamaoncol.2015.6053.

297. Penson RT, Sales E, Sullivan L, Borger DR, Krasner CN, Goodman AK, Del Carmen MG, Growdon WB, Schorge JO, Boruta DM, Castro CM, Dizon DS, **Birrer MJ**. A SNaPshot of potentially personalized care: Molecular diagnostics in gynecologic cancer. Gynecol Oncol. 2016 Apr;141(1):108-12. doi: 10.1016/j.ygyno.2016.02.032.

298. Myers AP, Filiaci VL, Zhang Y, Pearl M, Behbakht K, Makker V, Hanjani P, Zweizig S, Burke JJ 2nd, Downey G, Leslie KK, Van Hummelen P, **Birrer MJ**, Fleming GF. Tumor mutational analysis of GOG248, a phase II study of temsirolimus or temsirolimus and alternating megestrol acetate and tamoxifen for advanced endometrial cancer (EC): An NRG Oncology/Gynecologic Oncology Group study. Gynecol Oncol. 2016 Apr;141(1):43-8. doi: 10.1016/j.ygyno.2016.02.025.

299. Krzystyniak J, Ceppi L, Dizon DS, **Birrer MJ**. Epithelial ovarian cancer: the molecular genetics of epithelial ovarian cancer. Ann Oncol. 2016 Apr;27 Suppl 1:i4-i10. doi: 10.1093/annonc/mdw083.

300. Sosulski A, Horn H, Zhang L, Coletti C, Vathipadiekal V, Castro CM, **Birrer MJ**, Nagano O, Saya H, Lage K, Donahoe PK, Pépin D. CD44 Splice Variant v8-10 as a Marker of Serous Ovarian Cancer Prognosis. PLoS One. 2016 Jun 2;11(6):e0156595. doi: 10.1371/journal.pone.0156595. eCollection 2016.

301. Lax T, Dizon DS, **Birrer M**, Long A, Del Carmen M, Goodman A, Krasner C, Penson RT, Growdon W, Camargo CA Jr, Banerji A. Extended carboplatin infusion does not reduce frequency of hypersensitivity reaction at initiation of retreatment in patients with recurrent platinum-sensitive ovarian cancer. J Allergy Clin Immunol Pract. 2017 Jan - Feb;5(1):177-178. doi: 10.1016/j.jaip.2016.07.010. No abstract available.

302. Waldron L, Riester M, Ramos M, Parmigiani G, **Birrer M**. The Doppelgänger effect: Hidden duplicates in databases of transcriptome profiles. J Natl Cancer Inst. 2016 Jul 5;108(11). pii: djw146. doi: 10.1093/jnci/djw146.

303. Wilbur MB, **Birrer MJ**, Spriggs DR. CLINICAL DECISIONS. Chemotherapy-Induced Nausea and Vomiting. N Engl J Med. 2016 Jul 14;375(2):177-9. doi: 10.1056/NEJMclde1607577. No abstract available.

304. Rauh-Hain JA, **Birrer M**, Del Carmen MG. "Carcinosarcoma of the ovary, fallopian tube, and peritoneum: Prognostic factors and treatment modalities". Gynecol Oncol. 2016 Aug;142(2):248-54. doi: 10.1016/j.ygyno.2016.06.003.

305. Del Carmen MG, Supko JG, Horick NK, Rauh-Hain JA, Clark RM, Campos SM, Krasner CN, Atkinson T, **Birrer MJ**. Phase 1 and 2 study of carboplatin and pralatrexate in patients with recurrent, platinum-sensitive ovarian, fallopian tube, or primary peritoneal cancer. Cancer. 2016 Nov 15;122(21):3297-3306. doi: 10.1002/cncr.30196.

306. Liu JF, Moore KN, **Birrer MJ**, Berlin S, Matulonis UA, Infante JR, Wolpin B, Poon KA, Firestein R, Xu J, Kahn R, Wang Y, Wood K, Darbonne WC, Lackner MR, Kelley SK, Lu X, Choi YJ, Maslyar D, Humke EW, Burris HA. Phase I study of safety and pharmacokinetics of the anti-MUC16 antibody-drug conjugate DMUC5754A in patients with platinum-resistant ovarian cancer or unresectable pancreatic cancer. Ann Oncol. 2016 Nov;27(11):2124-2130.

307. Matulonis UA, Wulf GM, Barry WT, **Birrer M**, Westin SN, Farooq S, Bell-McGuinn KM, Obermayer E, Whalen C, Spagnoletti T, Luo W, Liu H, Hok RC, Aghajanian C, Solit DB, Mills GB, Taylor BS, Won H, Berger MF, Palakurthi S, Liu J, Cantley LC, Winer E. Phase I dose escalation study of the PI3kinase pathway inhibitor BKM120 and the oral poly (ADP ribose) polymerase (PARP) inhibitor olaparib for the treatment of high grade serous ovarian and breast cancer. Ann Oncol. 2016 Dec 19. pii: mdw672. doi: 10.1093/annonc/mdw672.

308. Moore KN, Martin LP, O'Malley DM, Matulonis UA, Konner JA, Perez RP, Bauer TM, Ruiz-Soto R, **Birrer MJ**. Safety and activity of mirvetuximab soravtansine (IMGN853), a folate receptor alpha-targeting antibody-drug conjugate, in platinum-resistant ovarian, fallopian tube, or primary peritoneal cancer: A phase I expansion study. J Clin Oncol. 2016 Dec 28:JCO2016699538.

309. McCourt CK, Deng W, Dizon DS, Lankes HA, **Birrer MJ**, Lomme MM, Powell MA, Kendrick JE, Saltzman JN, Warshal D, Tenney ME, Kushner DM, Aghajanian C. A phase II evaluation of ixabepilone in the treatment of recurrent/persistent carcinosarcoma of the uterus, an NRG Oncology/Gynecologic Oncology Group study. Gynecol Oncol. 2017 Jan;144(1):101-106. doi: 10.1016/j.ygyno.2016.10.026.

310. Cancer Genome Atlas Research Network. Integrated genomic and molecular characterization of cervical cancer. Nature. 2017 Jan 23. doi: 10.1038/nature21386.

311. Xiao H, Qi R, Li T, Awuah SG, Zheng Y, Wei W, Kang X, Song H, Wang Y, Yu Y, Bird MA, Jing X, Yaffe MB, **Birrer MJ**, Ghoroghchian PP. Maximizing Synergistic Activity When Combining RNAi and Platinum-based Anticancer Agents. J Am Chem Soc. 2017 Feb 6. doi: 10.1021/jacs.6b12108.

312. Gockley A, Melamed A, Bregar AJ, Clemmer JT, **Birrer M**, Schorge JO, Del Carmen MG, Rauh-Hain JA. Outcomes of women with high-grade and low-grade advanced-stage serous epithelial ovarian cancer. Obstet Gynecol. 2017 Mar;129(3):439-447. doi:

313. Yazinski SA, Comaills V, Buisson R, Genois MM, Nguyen HD, Ho CK, Todorova Kwan T, Morris R, Lauffer S, Nussenzweig A, Ramaswamy S, Benes CH, Haber DA, Maheswaran S, **Birrer MJ**, Zou L. ATR inhibition disrupts rewired homologous recombination and fork protection pathways in PARP inhibitor-resistant BRCA-deficient cancer cells. Genes Dev. 2017 Feb 1;31(3):318-332. doi: 10.1101/gad.290957.116. Epub 2017 Feb 27.

314. Phelan CM, Kuchenbaecker KB, Tyrer JP, Kar SP, Lawrenson K, Winham SJ, Dennis J, Pirie A, Riggan MJ, Chornokur G, Earp MA, Lyra PC Jr, Lee JM, Coetzee S, Beesley J, McGuffog L, Soucy P, Dicks E, Lee A, Barrowdale D, Lecarpentier J, Leslie G, Aalfs CM, Aben KK, Adams M, Adlard J, Andrulis IL, Anton-Culver H, Antonenkova N; AOCS study group., Aravantinos G, Arnold N, Arun BK, Arver B, Azzollini J, Balmaña J, Banerjee SN, Barjhoux L, Barkardottir RB, Bean Y, Beckmann MW, Beeghly-Fadiel A, Benitez J, Bermisheva M, Bernardini MQ, **Birrer MJ**, Bjorge L, Black A, Blankstein K, Blok MJ, Bodelon C, Bogdanova N, Bojesen A, Bonanni B, Borg Å, Bradbury AR, Brenton JD, Brewer C, Brinton L, Broberg P, Brooks-Wilson A, Bruinsma F, Brunet J, Buecher B, Butzow R, Buys SS, Caldes T, Caligo MA, Campbell I, Cannioto R, Carney ME, Cescon T, Chan SB, Chang-Claude J, Chanock S, Chen XQ, Chiew YE, Chiquette J, Chung WK, Claes KB, Conner T, Cook LS, Cook J, Cramer DW, Cunningham JM, D'Aloisio AA, Daly MB, Damiola F, Damirovna SD, Dansonka-Mieszkowska A, Dao F, Davidson R, DeFazio A, Delnatte C, Doheny KF, Diez O, Ding YC, Doherty JA, Domchek SM, Dorfling CM, Dörk T, Dossus L, Duran M, Dürst M, Dworniczak B, Eccles D, Edwards T, Eeles R, Eilber U, Ejlertsen B, Ekici AB, Ellis S, Elvira M; EMBRACE Study., Eng KH, Engel C, Evans DG, Fasching PA, Ferguson S, Ferrer SF, Flanagan JM, Fogarty ZC, Fortner RT, Fostira F, Foulkes WD, Fountzilas G, Fridley BL, Friebel TM, Friedman E, Frost D, Ganz PA, Garber J, García MJ, Garcia-Barberan V, Gehrig A; GEMO Study Collaborators., Gentry-Maharaj A, Gerdes AM, Giles GG, Glasspool R, Glendon G, Godwin AK, Goldgar DE, Goranova T, Gore M, Greene MH, Gronwald J, Gruber S, Hahnen E, Haiman CA, Håkansson N, Hamann U, Hansen TV, Harrington PA, Harris HR, Hauke J; HEBON Study., Hein A, Henderson A, Hildebrandt MA, Hillemanns P, Hodgson S, Høgdall CK, Høgdall E, Hogervorst FB, Holland H, Hooning MJ, Hosking K, Huang RY, Hulick PJ, Hung J, Hunter DJ, Huntsman DG, Huzarski T, Imyanitov EN, Isaacs C, Iversen ES, Izatt L, Izquierdo A, Jakubowska A, James P, Janavicius R, Jernetz M, Jensen A, Jensen UB, John EM, Johnatty S, Jones ME, Kannisto P, Karlan BY, Karnezis A, Kast K; KConFab Investigators., Kennedy CJ, Khusnutdinova E, Kiemeney LA, Kiiski JI, Kim SW, Kjaer SK, Köbel M, Kopperud RK, Kruse TA, Kupryjanczyk J, Kwong A, Laitman Y, Lambrechts D, Larrañaga N, Larson MC, Lazaro C, Le ND, Le Marchand L, Lee JW, Lele SB, Leminen A, Leroux D, Lester J, Lesueur F, Levine DA, Liang D, Liebrich C, Lilyquist J, Lipworth L, Lissowska J, Lu KH, Lubiński J, Luccarini C, Lundvall L, Mai PL, Mendoza-Fandiño G, Manoukian S, Massuger LF, May T, Mazoyer S, McAlpine JN, McGuire V, McLaughlin JR, McNeish I, Meijers-Heijboer H, Meindl A, Menon U, Mensenkamp AR, Merritt MA, Milne RL, Mitchell G, Modugno F, Moes-Sosnowska J, Moffitt M, Montagna M, Moysich KB, Mulligan AM, Musinsky J, Nathanson KL, Nedergaard L, Ness RB, Neuhausen SL, Nevanlinna H, Niederacher D, Nussbaum RL, Odunsi K, Olah E, Olopade OI, Olsson H, Olswold C, O'Malley DM, Ong KR, Onland-Moret NC; OPAL study group., Orr N, Orsulic S, Osorio A, Palli D, Papi L, Park-Simon TW, Paul J, Pearce CL, Pedersen IS, Peeters PH, Peissel B, Peixoto A, Pejovic T, Pelttari LM,

Permuth JB, Peterlongo P, Pezzani L, Pfeiler G, Phillips KA, Piedmonte M, Pike MC, Piskorz AM, Poblete SR, Pocza T, Poole EM, Poppe B, Porteous ME, Prieur F, Prokofyeva D, Pugh E, Pujana MA, Pujol P, Radice P, Rantala J, Rappaport-Fuerhauser C, Rennert G, Rhiem K, Rice P, Richardson A, Robson M, Rodriguez GC, Rodríguez-Antona C, Romm J, Rookus MA, Rossing MA, Rothstein JH, Rudolph A, Runnebaum IB, Salvesen HB, Sandler DP, Schoemaker MJ, Senter L, Setiawan VW, Severi G, Sharma P, Shelford T, Siddiqui N, Side LE, Sieh W, Singer CF, Sobol H, Song H, Southey MC, Spurdle AB, Stadler Z, Steinemann D, Stoppa-Lyonnet D, Sucheston-Campbell LE, Sukiennicki G, Sutphen R, Sutter C, Swerdlow AJ, Szabo CI, Szafron L, Tan YY, Taylor JA, Tea MK, Teixeira MR, Teo SH, Terry KL, Thompson PJ, Thomsen LC, Thull DL, Tihomirova L, Tinker AV, Tischkowitz M, Tognazzo S, Toland AE, Tone A, Trabert B, Travis RC, Trichopoulou A, Tung N, Tworoger SS, van Altena AM, Van Den Berg D, van der Hout AH, van der Luijt RB, Van Heetvelde M, Van Nieuwenhuysen E, van Rensburg EJ, Vanderstichele A, Varon-Mateeva R, Vega A, Edwards DV, Vergote I, Vierkant RA, Vijai J, Vratimos A, Walker L, Walsh C, Wand D, Wang-Gohrke S, Wappenschmidt B, Webb PM, Weinberg CR, Weitzel JN, Wentzensen N, Whittemore AS, Wijnen JT, Wilkens LR, Wolk A, Woo M, Wu X, Wu AH, Yang H, Yannoukakos D, Ziogas A, Zorn KK, Narod SA, Easton DF, Amos CI, Schildkraut JM, Ramus SJ, Ottini L, Goodman MT, Park SK, Kelemen LE, Risch HA, Thomassen M, Offit K, Simard J, Schmutzler RK, Hazelett D, Monteiro AN, Couch FJ, Berchuck A, Chenevix-Trench G, Goode EL, Sellers TA, Gayther SA, Antoniou AC, Pharoah PD. Identification of 12 new susceptibility loci for different histotypes of epithelial ovarian cancer. Nat Genet. 2017 Mar 27. doi: 10.1038/ng.3826.

315. Moore KN, Borghaei H, O'Malley DM, Jeong W, Seward SM, Bauer TM, Perez RP, Matulonis UA, Running KL, Zhang X, Ponte JF, Ruiz-Soto R, **Birrer MJ**. Phase 1 dose-excalation study of mirvetuximab soravtansine (IMGN853), a folate receptor α-targeting antibody-drug conjugate, in patients with solid tumors. Cancer 2017 Apr 25. doi: 10.1002/cncr.30736.

316. Tao Z, Dang X, Huang X, Muzumdar MD, Xu ES, Neelkanth NM, Song H, Qi R, Yu Y, Li T, Wei W, Wyckoff J, **Birrer MJ**, Belcher AM, Ghoroghchian PP. Early tumor detection afforded by *in vivo* imaging of near infrared II flourescence. Biomaterials 134 July 2017, 202-215 doi: 10.1016/j.biomaterials. 2017.04.046.

317. Reyes HD, Miecznikowski J, Gonzalez-Bosquet J, Devor EJ, Zhang Y, Thiel KW, Samuelson MI, McDonald M, stephan JM, Hanjani P, Guntupalli S, Tewari KS, Backes F, Ramirez N, Fleming GF, Filiaci V, **Birrer MJ**, Leslie KK. High stathmin expression is a marker for poor clinical outcome in endometrial cancer: An NRG oncology group/gynecologic oncology group study. Gynecol Oncol. 2017, May 19 pii: S0090-8258(17)30870-3. Doi: 10.1016/j.ygyno.2017.05.017.

318. Tewari KS, Sill MW, Penson RT, Huang H, Ramondetta LM, Landrum LM, Oaknin A, Reid TJ, Leitao MM, Michael HE, DiSaia PJ, Copeland LJ, Creasman WT, Stehman FB, Brady MF, Burger RA, Thigpen JT, **Birrer MJ**, Waggoner SE, Moore DH, Look KY, Koh WJ, Monk BJ. Bevacizumab for advanced cervical cancer: final overall survival and adverse event analysis of a randomised, controlled, open-label, phase 3 trial (Gynecologic Oncology Group 240). Lancet. 2017 Jul 27. pii: S0140-6736(17)31607-0. doi: 10.1016/S0140-6736(17)31607-0.

319. Martin LP, Konner JA, Moore KN, Seward SM, Matulonis UA, Perez RP, Su Y, Berkenblit A, Ruiz-Soto R, **Birrer MJ**. Characterization of folate receptor alpha (FRα) expression in archival

tumor and biopsy samples from relapsed epithelial ovarian cancer patients: A phase I expansion study of the FRα-targeting antibody-drug conjugate mirvetuximab soravtansine. Gynecol Oncol. 2017 Aug 24. pii: S0090-8258(17)31253-2. doi: 10.1016/j.ygyno.2017.08.015.

320. Moore KN, Tritchler D, Kaufman KM, Lankes H, Quinn MCJ; Ovarian Cancer Association Consortium, Van Le L, Berchuck A, Backes FJ, Tewari KS, Lee RB, Kesterson JP, Wenham RM, Armstrong DK, Krivak TC, Bookman MA, **Birrer MJ**. Genome-wide association study evaluating single-nucleotide polymorphisms and outcomes in patients with advanced stage serous ovarian or primary peritoneal cancer: An NRG Oncology/Gynecologic Oncology Group study. Gynecol Oncol. 2017 Sep 18. pii: S0090-8258(17)31261-1. doi: 10.1016/j.ygyno.2017.08.024.

321. Devor EJ, Miecznikowski J, Schickling BM, Gonzalez-Bosquet J, Lankes HA, Thaker P, Argenta PA, Pearl ML, Zweizig SL, Mannel RS, Brown A, Ramirez NC, Ioffe OB, Park KJ, Creasman WT, **Birrer MJ**, Mutch D, Leslie KK. Dysregulation of miR-181c expression influences recurrence of endometrial endometrioid adenocarcinoma by modulating NOTCH2 expression: An NRG Oncology/Gynecologic Oncology Group study. Gynecol Oncol. 2017 Sep 29. pii: S0090-8258(17)31349-5. doi: 10.1016/j.ygyno.2017.09.025.

322. Bais C, Mueller B, Brady MF, Mannel RS, Burger RA, Wei W, Marien KM, Kockx MM, Husain A, **Birrer MJ**; Tumor Microvessel Density as a Potential Predictive Marker for Bevacizumab Benefit: GOG-0218 Biomarker Analyses. NRG Oncology/Gynecologic Oncology Group. J Natl Cancer Inst. 2017 Nov 1;109(11). doi: 10.1093/jnci/djx066.

323. Tsang TY, Wei W, Itamochi H, Tambouret R, **Birrer MJ**. Integrated genomic analysis of clear cell ovarian cancers identified PRKCI as a potential therapeutic target. Oncotarget. 2017 Aug 4;8(57):96482-96495. doi: 10.18632/oncotarget.19946. eCollection 2017 Nov 14. PMID: 29228547

324. Moore KN, Martin LP, O'Malley DM, Matulonis UA, Konner JA, Vergote I, Ponte JF, **Birrer MJ.** A review of mirvetuximab soravtansine in the treatment of platinum-resistant ovarian cancer. Future Oncol. 2018 Jan;14(2):123-136. doi: 10.2217/fon-2017-0379. Epub 2017 Nov 3.

325. Norquist BM, Brady MF, Harrell MI, Walsh T, Lee MK, Gulsuner S, Bernards SS, Casadei S, Burger RA, Tewari KS, Backes F, Mannel RS, Glaser G, Bailey C, Rubin S, Soper J, Lankes HA, Ramirez NC, King MC, **Birrer MJ**, Swisher EM. Mutations in Homologous Recombination Genes and Outcomes in Ovarian Carcinoma Patients in GOG 218: An NRG Oncology/Gynecologic Oncology Group Study. Clin Cancer Res. 2017 Nov 30. doi: 10.1158/1078-0432.CCR-17-1327.

326. Noh K, Mangala LS, Han HD, Zhang N, Pradeep S, Wu SY, Ma S, Mora E, Rupaimoole R, Jiang D, Wen Y, Shahzad MMK, Lyons Y, Cho M, Hu W, Nagaraja AS, Haemmerle M, Mak CSL, Chen X, Gharpure KM, Deng H, Xiong W, Kingsley CV, Liu J, Jennings N, **Birrer MJ,** Bouchard RR, Lopez-Berestein G, Coleman RL, An Z, Sood AK. Differential Effects of EGFL6 on Tumor versus Wound Angiogenesis. Cell Rep. 2017 Dec 5;21(10):2785-2795. doi: 10.1016/j.celrep.2017.11.020.

327. Tsang TY, Wei W, Itamochi H, Tambouret R, **Birrer MJ.** Integrated genomic analysis of clear cell ovarian cancers identified PRKCI as a potential therapeutic target. Oncotarget. 2017 Aug 4;8(57):96482-96495. doi: 10.18632/oncotarget.19946. eCollection 2017 Nov 14.

328. Wei W, Giulia F, Luffer S, Kumar R, Wu B, Tavallai M, Bekele RT, **Birrer MJ.** How can molecular abnormalities influence our clinical approach? Ann Oncol. 2017 Nov 1;28(suppl_8):viii16-viii24. doi: 10.1093/annonc/mdx447.

329. Moore KN, Martin LP, O'Malley DM, Matulonis UA, Konner JA, Vergote I, Ponte JF, **Birrer MJ**. A review of mirvetuximab soravtansine in the treatment of platinum-resistant ovarian cancer. Future Oncol. 2018 Jan;14(2):123-136. doi: 10.2217/fon-2017-0379. Epub 2017 Nov 3. PMID: 29098867

330. Leung CS, Yeung TL, Yip KP, Wong KK, Ho SY, Mangala LS, Sood AK, Lopez-Berestein G, Sheng J, Wong ST, **Birrer MJ**, Mok SC. Cancer-associated fibroblasts regulate endothelial adhesion protein LPP to promote ovarian cancer chemoresistance. J Clin Invest. 2018 Feb 1;128(2):589-606. doi: 10.1172/JCI95200. Epub 2017 Dec 18. PMID: 29251630

331. Qi R, Wang Y, Bruno PM, Xiao H, Yu Y, Li T, Lauffer S, Wei W, Chen Q, Kang X, Song H, Yang X, Huang X, Detappe A, Matulonis U, Pepin D, Hemann MT, **Birrer MJ**, Ghoroghchian PP. Nanoparticle conjugates of a highly potent toxin enhance safety and circumvent platinum resistance in ovarian cancer. Nat Commun. 2017 Dec 18;8(1):2166. doi: 10.1038/s41467-017-02390-7. Erratum in: Nat Commun. 2018 Feb 7;9(1):628. PMID: 29255160

332. Moore KN, Vergote I, Oaknin A, Colombo N, Banerjee S, Oza A, Pautier P, Malek K, **Birrer MJ**. FORWARD I: a Phase III study of mirvetuximab soravtansine versus chemotherapy in platinum-resistant ovarian cancer. Future Oncol. 2018 Feb 9. doi: 10.2217/fon-2017-0646. [Epub ahead of print] PMID: 29424243

333. Hamilton CA, Miller A, Casablanca Y, Horowitz NS, Rungruang B, Krivak TC, Richard SD, Rodriguez N, **Birrer MJ**, Backes FJ, Geller MA, Quinn M, Goodheart MJ, Mutch DG, Kavanagh JJ, Maxwell GL, Bookman MA. Clinicopathologic characteristics associated with long-term survival in advanced epithelial ovarian cancer: an NRG Oncology/Gynecologic Oncology Group ancillary data study. Gynecol Oncol. 2018 Feb;148(2):275-280. doi: 10.1016/j.ygyno.2017.11.018.

334. Banerjee S, Oza AM, **Birrer MJ**, Hamilton EP, Hasan J, Leary A, Moore KN, Mackowiak-Matejczyk B, Pikiel J, Ray-Coquard I, Trask P, Lin K, Schuth E, Vaze A, Choi Y, Marsters JC, Maslyar DJ, Lemahieu V, Wang Y, Humke EW, Liu JF. Anti-NaPi2b antibody-drug conjugate lifastuzumab vedotin (DNIB0600A) compared with pegylated liposomal doxorubicin in patients with platinum-resistant ovarian cancer in a randomized, open-label, phase II study. Ann Oncol. 2018 Apr 1;29(4):917-923. doi: 10.1093/annonc/mdy023. PMID: 29401246

335. Norquist BM, Brady MF, Harrell MI, Walsh T, Lee MK, Gulsuner S, Bernards SS, Casadei S, Burger RA, Tewari KS, Backes F, Mannel RS, Glaser G, Bailey C, Rubin S, Soper J, Lankes HA, Ramirez NC, King MC, **Birrer MJ**, Swisher EM. Mutations in Homologous Recombination Genes and Outcomes in Ovarian Carcinoma Patients in GOG 218: An NRG Oncology/Gynecologic Oncology Group Study. Clin Cancer Res. 2018 Feb 15;24(4):777-783. doi: 10.1158/1078-0432.CCR-17-1327. Epub 2017 Nov 30. PMID: 29191972

336. Moore KN, **Birrer MJ**. Administration of the Tablet Formulation of Olaparib in Patients with Ovarian Cancer: Practical Guidance and Expectations. Oncologist. 2018 Mar 28. pii:

theoncologist.2017-0485. doi: 10.1634/theoncologist.2017-0485. [Epub ahead of print] Review. PMID: 29593098

337. Caumanns JJ, Berns K, Wisman GBA, Fehrmann RSN, Tomar T, Klip H, Meersma GJ, Hijmans EM, Gennissen A, Duiker EW, Weening D, Itamochi H, Kluin RJ, Reyners AKL, **Birrer MJ**, Salvesen HB, Vergote I, Van Nieuwenhuysen E, Brenton JD, Braicu EI, Kupryjanczyk J, Spiewankiewicz B, Mittempergher L, Bernards R, van der Zee AGJ, de Jong S. Integrative kinome profiling identifies mTORC1/2 inhibition as treatment strategy in ovarian clear cell carcinoma. Clin Cancer Res. 2018 Apr 23. pii: clincanres.3060.2017. doi: 10.1158/1078-0432.CCR-17-3060. [Epub ahead of print] PMID: 29685880

338. Moore KN, O'Malley DM, Vergote I, Martin LP, Gonzalez-Martin A, Malek K, **Birrer MJ**. Safety and activity findings from a phase 1b escalation study of mirvetuximab soravtansine, a folate receptor alpha (FRα)-targeting antibody-drug conjugate (ADC), in combination with carboplatin in patients with platinum-sensitive ovarian cancer. Gynecol Oncol. 2018 Oct; 151(1):46-52. doi: 10.1016/j.ygyno.2018.07.017. Epub 2018 Aug 6. PMID: 30093227

339. Matulonis UA, **Birrer MJ**, O'Malley DM, Moore KN, Konner J, Gilbert L, Martin LP, Bauer TM, Oza AM, Malek K, Pinkas J, Kim SK. Evaluation of Prophylactic Corticosteroid Eye Drop Use in the Management of Corneal Abnormalities Induced by the Antibody-Drug Conjugate Mirvetuximab Soravtansine. Clin Cancer Res. 2018 Nov 9. doi: 10.1158/1078-0432.CCR-18-2474. [Epub ahead of print] PMID: 30413525

340. Yeung TL, Tsai CC, Leung CS, Au Yeung CL, Thompson MS, Lu KH, Freedman RS, **Birrer MJ**, Wong KK, Mok SC. ISG15 Promotes ERK1 ISGylation, CD8+ T Cell Activation and Suppresses Ovarian Cancer Progression. Cancers (Basel). 2018 Nov 22;10(12). pii: E464. doi: 10.3390/cancers10120464. PMID: 30469497. PMCID: PMC6316352.

341. Rauh-Hain JA, Hidrue MK, Gaccione P, Melamed A, Meyer LA, Keating NL, Giordano SH, Rice LW, **Birrer MJ**, Del Carmen MG. Variation in resource utilization associated with the surgical management of ovarian cancer. Gynecol Oncol. 2018 Dec 19. pii: S0090-8258(18)31513-0. doi: 10.1016/j.ygyno.2018.12.013. [Epub ahead of print] PMID: 30579568.

342. **Birrer MJ**, Betella I, Martin LP, Moore KN. Is Targeting the Folate Receptor in Ovarian Cancer Coming of Age? Oncologist. 2019 Jan 11. pii: theoncologist.2018-0459. doi: 10.1634/theoncologist.2018-0459. [Epub ahead of print] No abstract available. PMID: 30635448

BOOKS AND BOOK CHAPTERS

1. Minna JD, Battey JF, **Birrer MJ**, Cuttitta F, DeGreve J, Gazdar AF, Ihde DC, Johnson BE, Kaye F, Krystal GW, Lebacq AM, Linnoila IR, Mulshine J, Nau MM, Sausville EA, Schutte T, Trepel JB. Genetic changes involved in the pathogenesis of human lung cancer including oncogene activation, chromosomal deletions, and autocrine growth factor production. In: Fortner JG, Rhoads JE, editors. Accomplishments in cancer research. Philadelphia: JB Lippincott, 1988. p. 155-182.

2. Minna JD, Kaye FJ, Takahashi T, Harbour JW, Rosenberg R, Nau M, Whang-Peng J, Johnson B,

Birrer MJ, Gazdar AF. Recessive oncogenes and chromosomal deletions in human lung cancer. In: Cavennee W, Hastie N, Stanbridge E, editors. Recessive oncogenes and tumor suppression. Cold Spring Harbor Laboratory, 1989, p. 57-65.

3. Brown PH, Mulshine J, and **Birrer MJ**. Tumor initiators and promoters in lung cancer. Lung Cancer Research Quarterly 1:3-16, 1991.

4. Siegfried JM, **Birrer MJ**, and Cuttitta F. Detection of growth factor effects and expression in normal and neoplastic human bronchial epithelial cell. In: Milo G.E., et al. editors. Transformation of Human Epithelial Cells: Molecular Oncogenetic Mechanisms. Boca Raton: CRC, 1992.

5. Mulshine JL, **Birrer MJ**, Treston AM, Scott F, Quinn K, Avis I, and Cuttitta F: Growth factors and targets for rational application intervention agents. In: Newell GR, Hong WK, editors. The Biology and Prevention of Aerodigestive Tract Cancers. New York: Plenum, 1992. p. 81-88.

6. Mulshine JL, Shaw GL, Cuttitta F, Scott F, Avis I, Treston AM, Linnoila RI, **Birrer MJ**, Brown P, Gupta PK, and Tockman MS: Applicatons of biomakers for lung cancer early detection. In Fortner JG. Rhoads JE, editors. Accomplishments in cancer research. Philadelphia: JB Lippincott, 1992. p. 204-218.

7. Draoui M, Battey J, **Birrer MJ**, Moody TW. Regulation of gastrin releasing peptide gene expression. In: Moody TW, editor. Growth Factors, Peptides, and Receptors. New York: Plenum, 1993. p. 347-352.

8. Teneriello MG, Hall KL, Lemon SJ, Nash JD, **Birrer MJ**. Genetic alterations in gynecologic cancers. In: Srivastava S, Lippman SM, Hong WK, Mulshine, JL, editors. Early Detection of Cancer: Molecular Markers. Armonk, New York: Futura Publishing, 1994. p. 97-119.

9. **Birrer MJ**, Gray JW, Lippman SM, Lubet R, Mulshine JL, Shaw GL, Srivastava S, and Tockman S. Basic science, technical development, and clinical and laboratory-to-field application issues. In: Srivastava S, Lippman SM, Hong WK, Mulshine JL, editors. Early Detection of Cancer: Molecular Markers. Armonk, New York: Futura, 1994. p. 289-308.

10. Szabo E, **Birrer, MJ**. Oncogenes, cancer and growth factors. In: Barnes PJ and Stockley RA, editors. Molecular biology of lung diseases. Oxford: Blackwell Scientific, 1994. p. 173-191.

11. Lemon SJ, Sabichi AL, and **Birrer MJ**. Genetic alterations in lung cancer. In: Garrett CT and Sells S, editors. Cellular Cancer Markers. Totowa: Humana, 1995. p. 209-231.

12. Sabichi AL, Bober MA, and **Birrer MJ**. Lung cancer. In Kurzrock R and Talpaz M, editors. Molecular biology in cancer medicine. London: Martin Dunitz, 1995. p. 223-241.

13. Parker MF, Sausville EA, **Birrer MJ**. Basic biology and biochemistry of gynecologic cancer. In: Hoskins W, Perez CA, Young RC, editors. Principles and practice of gynecology oncology, 2nd edition. Philadelphia: Lippincott-Raven, 1997, p. 61-86.

14. Farley J, Hendricks D, **Birrer MJ**. Ovarian and endometrial cancer molecular pathology. In: Srivastava S, Henson DE, and Gazdar A, editors. Molecular pathology of early cancer. Amsterdam, IOS, 1998, p.21-34.

15. Rose GS, Carlson J, and **Birrer MJ**. Basic biology and biochemistry of gynecologic cancer. In: Hoskins, W, Perez, CA, and Young, RC, editors. Principles and practice of gynecology oncology, 2nd ed. Philadelphia: Lippincott-Raven, 2000. p. 62-85.

16. Zorn K, Gardner G, **Birrer MJ**. Basic biology of gynecologic cancer. In: Hoskins W, Perez CA, Young RC, editors. Principles and practice of gynecology oncology, 3rd edition. Philadelphia: Lippincott-Raven, 2004.

17. Zorn KK, Gardner GJ, **Birrer MJ**. Epithelial ovarian Cancer. In: Kelloff GJ, Hawk ET, Sigman CC, editors. Cancer Chemoprevention Volume 2: Strategies for cancer chemoprevention. Totowa, Humana Press Inc, 2005. p. 473-489.

18. Annunziata CM, and **Birrer, MJ**. Endometrial Cancer. In: Abraham J, Allegra CJ, Gulley J, editors. Bethesda Handbook of Clinical Oncology, 2nd ed. Philadelphia, Lippincott Williams & Wilkins, 2005. p. 237-245.

19. Annunziata CM, and **Birrer, MJ**. Vulvar Cancer. In: Abraham J, Allegra CJ, Gulley J, editors. Bethesda. Handbook of Clinical Oncology, 2nd edition. Philadelphia, Lippincott Williams & Wilkins, 2005. p. 257-265.

20. Zorn K, Gardner G; Farley J, and **Birrer, MJ**. Basic biology of gynecologic cancer. In: Hoskins W, Perez CA, Young RC, editors. Principles and practice of gynecology oncology, 4th edition. Philadelphia, Lippincott-Raven, 2007.

21. **Birrer MJ**, Zorn, KK, Gardner, GJ. Molecular profiling in ovarian cancer. In: Levenback CF, Sood, AK, Lu KH, Coleman RL, editors. Prognostic and predictive factors in gynecologic cancers. London, UK: Informa Ltd, 2007.

22. Farley J, Ozbun, LL, **Birrer MJ**. Whole-genome expression profiling of papillary serous ovarian cancer: Activted pathways, potential targets and noise. In: Yuryev A, editor. A pathway analysis for drug discovery. Hoboken, NJ: John Wiley & Sons, Inc., 2008.

23. Stany M, Farley J, **Birrer MJ**. Chemotherapy: principles and practice. In: Fishman D, editor. Gynecologic oncology. Requisites in obstetrics and gynecology, Philadelphia: Elsevier-Global Medicine, 2008.

24. Farley J, **Birrer MJ**. Novel therapeutic targets. In: Stack MS, Fishman DA, editors. Ovarian cancer, 2nd edition. Springer Science Business Media, LLS, 2009. p. 63-84.

25. Annunziata CM, **Birrer MJ**. Vulvar cancer. In: Abraham J, Allegra C, Gulley J, editors. Bethesda Manual of Clinical Oncology. Philadelphia: Lippincott, Williams and Wilkins, 2008. p. 257-264. (3rd ed. 2010, p. 252-260)

26. Annunziata CM, **Birrer MJ**. Endometrial Carcinoma. In: Abraham J, Allegra C, Gulley J, editors. Bethesda Manual of Clinical Oncology. Philadelphia: Lippincott, Williams and Wilkins, 2008, p. 237-244. (3rd ed. 2010, p. 233-240)

27. McNally T, Penson R, Tran C, **Birrer MJ**. Gynecologic Cancer. In: Skeel RT, Khlief SN, editors. Handbook of Cancer Chemotherapy. 8th Edition. Philadelphia: Lippincott, Williams and Wilkins, 2011. p. 199-232.

28. Pejovic T, **Birrer MJ**, Anderson ML, Odunsi K. Molecular biology of gynecologic cancers. In: DeVita VT, Lawrence TS, Rosenberg SA, editors. Cancer: Principles and practice of oncology. 10th edition. Philadelphia: Lippincott, Williams and Wilkins, Jan 2015. p. 1005-1012.

29. del Carmen MG, Young RH, Schorge JO, **Birrer MJ**, editors. Uncommon Gynecologic Cancers. John Wiley & Sons, Ltd., 2015

30. **Birrer M**, Ceppi L, editors. Translational Advances in Gynecologic Cancers. Elsevier, 2017.

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APPENDIX B

Fed. R. Civ. P. 26(a)(2)(B)(v) Disclosure for Michael Birrer, MD, PhD

(As of Feb. 2019)

Depositions

- *Blaes v. Johnson & Johnson*, No. 1422-CC09326-01 (Mo. Cir. Ct. deposed June 8-9, 2017)

- *Brower v. Johnson & Johnson*, No. 16-EV-005534 (Ga. Fulton Cnty. deposed Sept. 25, 2018)